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Appendix A to RI Report

Endangerment Assessment Report

Nease Chemical Company – Salem, Ohio Site

Volume 2 of 4

Submitted to

United States Environmental Protection Agency

Region 5, Chicago, Illinois

and

Ohio Environmental Protection Agency

Columbus, Ohio

July 6, 1993: Revision 1
April 5, 1991: Original Submittal

Submitted by

Ruetgers-Nease Chemical Company, Inc.

State College, Pennsylvania

ENDANGERMENT ASSESSMENT FOR THE NEASE CHEMICAL COMPANY SALEM, OHIO SITE

Prepared for

Ruetgers-Nease Chemical Company, Inc.
State College, Pennsylvania

Prepared by

ENVIRON Corporation
Arlington, Virginia

July 2, 1993

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I. INTRODUCTION

A. Background

Through a corporate acquisition, Ruetgers-Nease Chemical Company, Inc. (Ruetgers-Nease) owns a former chemical manufacturing plant site in Salem, Ohio (the "Site"). In 1983, the Site was placed on the National Priorities List (NPL). A number of studies have been conducted to characterize the Site and the surrounding area. Most recently, ERM-Midwest, Inc. (ERM) conducted a Remedial Investigation (RI) of the Site. Field investigation activities conducted as part of the RI included geophysical investigations, monitor well drilling and installation, aquifer testing, a residential well survey, and topographic mapping and surveying. In addition, to determine the nature, extent, and magnitude of chemicals present in various environmental media at the Site and surrounding area, ERM collected and analyzed samples from on-site ambient air, off-site soil borings, on-site and off-site test pits, on-site pond borings, on-site surface water, and on-site and off-site ground water wells. In addition, ERM completed a sampling program for the Middle Fork of Little Beaver Creek (MFLBC), the main surface water body receiving runoff from the Site. This program included the analysis of samples collected from surface water, stream sediment, flood plain soil, and fish tissue at locations along the MFLBC from upstream of the Site to near East Liverpool, Ohio. A full description of the field investigation activities and sampling program is presented in the RI report prepared by ERM (1993).

ENVIRON Corporation (ENVIRON) was retained by Ruetgers-Nease to prepare an Endangerment Assessment (EA) for the Site[1], adjacent off-site areas, and the MFLBC. As indicated in the U.S. Environmental Protection Agency's (USEPA) *Risk Assessment Guidance for Superfund* ("RAGS", p. 1-4; USEPA 1989a), the objective of the EA, also referred to as the baseline risk assessment, is to characterize the potential risks to public health and the environment associated with hazardous substance releases from a site in the absence of any actions to control or mitigate these releases (i.e., under an assumption of no action).

[1] The Site is defined here as all areas within the Ruetgers-Nease property boundary.

B. The Risk Assessment Process

The assessment of potential risks described in this document is based on guidelines provided by the USEPA, and in particular RAGS (USEPA 1989a, 1989b). The foundation for this guidance comes from established chemical risk assessment principles and procedures developed for the regulation of environmental contaminants (NRC 1983; OSTP 1985) and other USEPA guidelines (e.g., USEPA 1986a).

Application of these guidelines and principles has provided a consistent process for evaluating and documenting potential health risks associated with environmental exposures. As emphasized by the Office of Science and Technology Policy (OSTP 1985) and USEPA (1986a) with respect to carcinogenic risk assessments, these assessments also involve a number of assumptions and forms of extrapolation that have not been verified by traditional scientific means. This approach has arisen because of the need, as perceived by regulatory officials, to act in the absence of complete experimental information by adopting a series of conservative assumptions to ensure maximum health protection. Risk assessments performed in this manner are designed to place an upper bound on risk. Similarly, risk assessment methods developed for the noncarcinogenic effects of chemicals incorporate various conservative (i.e., health protective) assumptions. Noncarcinogenic risk assessment is not intended to provide a demarcation between "safe" and "unsafe" levels of exposure. A substantial margin of safety is built into noncarcinogenic toxicity values[2], thereby providing a high degree of certainty that the levels derived as "acceptable" according to methods developed by regulatory agencies will cause no adverse health effects in the potentially exposed population. Consequently, exposures may even exceed the acceptable dose level without a significant risk arising.

It must be emphasized that the potential risks estimated using these risk assessment methods are not actuarial, i.e., the risk estimates cannot be used to predict the actual number of individuals who might experience health consequences as a result of exposure. Actual health risk is almost certainly less than that described using the methods of risk assessment. Furthermore, the risk estimates developed herein do not relate to absolute individual risks. Many individual risk factors -- such as exposures to other environmental agents, occupational exposures, smoking, age, diet, and inherent susceptibility -- will influence the probability of developing a specific disease.

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[2] Noncarcinogenic toxicity values are referred to by USEPA as reference doses (RfDs). The term RfD is further described in Chapter IV, Toxicological Assessment.

Although current risk assessment approaches generally overstate risk, they nevertheless provide a systematic approach that allows public health policy makers to establish the relative risk posed by various environmental substances and potential exposure pathways. A further discussion of uncertainties in the risk assessment process and the conservative assumptions adopted in light of this uncertainty is presented in Chapter VIII, Risk Characterization.

C. Report Organization

This report, which has been prepared in accordance with USEPA risk assessment guidance (USEPA 1989a, 1989b), is divided into ten chapters. Chapters I through VIII deal primarily with the public health risk assessment, while chapters IX and X deal with the environmental or ecological risk assessment. These chapters are as follows:

Chapter I. Introduction, in which background on the project, a discussion of the risk assessment process, and the report organization are presented.

Chapter II. Site Description and History, in which the description and history of the Site relevant to the assessment of human health and environmental risks are summarized.

Chapter III. Development of a Set of Chemical Data for Use in the Endangerment Assessment, in which chemicals are selected to focus the assessment on those chemicals that are most likely to pose the greatest potential public health risk.

Chapter IV. Toxicological Assessment, in which the hazard identification and dose-response evaluation for each selected chemical are completed to derive toxicity values that can be used to estimate the potential for adverse effects occurring in humans at different exposure levels.

Chapter V. Identification of Exposure Pathways, in which potential exposure pathways under current and hypothetical future conditions at the Site, adjacent off-site areas, and the area along the MFLBC are identified.

Chapter VI. Estimation of Environmental Concentrations, in which chemical concentrations are estimated for the various environmental media associated with the potential exposure pathways.

Chapter VII. Estimation of Human Intake, in which estimates of chemical concentrations at the points of human exposure are combined with exposure assumptions (e.g., the duration of exposure, the amount of chemical absorbed in the body, and the characteristics of the population receiving the exposure) to arrive at estimates of human intake or dose.

Chapter VIII. Risk Characterization, in which numerical estimates of carcinogenic and noncarcinogenic risks are calculated for each chemical by each potential route of exposure using the toxicity information and the estimates of human intake.

Chapter IX. Off-site Ecological Risk Assessment, in which the principles of risk assessment are used to evaluate the potential effects on the off-site local flora and fauna.

Chapter X. On-site Ecological Risk Assessment, in which the principles of risk assessment are used to evaluate the potential effects on the on-site local flora and fauna.

In addition, a number of technical appendices to the report provide the necessary documentation of data and methods relied upon to perform the analyses.

The environmental data contained in this EA are based solely on air, surface water, ground water, sediment, soil, and fish tissue sampling results presented in the RI report (ERM 1993), residue data for game collected by the Ohio Department of Health (ODH 1990), milk and meat residue data collected by the Ohio Department of Agriculture, a quantitative fish survey conducted by the Ohio Environmental Protection Agency (OEPA 1985), and a benthic macroinvertebrate survey conducted by Metcalf & Eddy (1988). In addition, results of other studies are incorporated, including the natural history of eastern Ohio to identify mammals, birds, and plants typical of the region, various listings of rare, threatened, and endangered species to determine their likely occurrence in the study area, inventory reports to identify wetlands areas, and characterizations of the extent of the MFLBC concerning wild and scenic river status.

II. SITE DESCRIPTION AND HISTORY

A. Introduction

This chapter summarizes those portions of the Site description and history that are relevant to the assessment of the human health risks. A more detailed description and history of the Site is presented in Chapter I of the RI report (ERM 1993).

B. Site Description

The Site is located in northeastern Ohio in a rural area northwest of the City of Salem (see Figure 1). The Site is approximately 44 acres, is surrounded mostly by residential and farm land, and has an industrial plant to the east and northeast (Crane-Deming). The MFLBC, located less than 1500 feet from the Site, is the principal surface water body receiving runoff from the Site. The MFLBC originates near the Site in Salem and is connected with the property via Feeder Creek. From Salem, the MFLBC flows north for about five miles, turns and flows eastward and then southeastward through Lisbon, Ohio, and eventually joins other tributaries to become Little Beaver Creek. Little Beaver Creek flows into the Ohio River near East Liverpool, Ohio.

The Site hydrogeology is presented in Section 3.7.2 of the RI report (ERM 1993). In summary, glacial materials at the Site are primarily composed of till, sand, and lake clays. The till and lake clays act as aquitards that separate the seven sand bodies identified at the Site into individual hydrogeologic units. These units are termed Sands 1 through 7. The primary bedrock units of interest at the Site are the Middle Kittanning Sandstone and the Vanport Limestone/Putnam Hill Shale Zone. These two bedrock units are separated by the Columbiana Shale, which acts as an aquitard.

C. Site History

From January 1961 until December 1973, a chemical manufacturing plant operated at the Site. During this period, Nease Chemical Company (Nease), which owned the Site, manufactured a variety of specialty chemicals including household cleaning compounds, fire retardants, pesticides, and chemical intermediates used in agricultural, pharmaceutical, and other chemical products. Products and chemical intermediates were manufactured in batch processes, and finished products were stored in warehouses, bulk storage, and tanks. Wastes

generated during the production of chemicals were neutralized and treated in five on-site ponds. Effluent from the ponds was discharged to the Salem Wastewater Treatment Plant from the late 1960s to 1975.

Manufacturing operations at the Site were discontinued in 1973. The majority of the buildings and manufacturing equipment on the Site were removed during decommissioning activities. As of December 30, 1977, Nease's assets (including the vacant Site) were acquired and merged with Ruetgers Chemicals, Inc. The new company resulting from the merger was Ruetgers-Nease Chemical Company, Inc.

Environmental investigations at the Site and surrounding area began in 1982 at the request of the Ohio Environmental Protection Agency (OEPA). A history of environmental work conducted from 1982 to 1988 is presented in Section 1.2.1 of the RI (ERM 1993). In January 1988, an Administrative Order of Consent (CO) was signed by Ruetgers-Nease and the USEPA and the OEPA, which required Ruetgers-Nease to conduct a RI and Feasibility Study (FS) in accordance with Section 121 of CERCLA. A work plan that presented the scope of work for the RI was approved by the Agencies on March 20, 1990, and work was initiated by ERM, Ruetgers-Nease's selected contractor, in April 1990.

III. DEVELOPMENT OF A SET OF CHEMICAL DATA FOR USE IN THE ENDANGERMENT ASSESSMENT

A. Sampling Data and Data Validation

ERM conducted site characterization work on behalf of Ruetgers-Nease as part of the RI. The design and implementation of these investigative studies have been approved by USEPA Region V. This work forms the basis for evaluating potential exposures to chemicals detected at the Site.

Both ENSECO, Inc. and Midwest Research Institute Laboratories analyzed the samples and sent the resulting data to Environmental Standards, Inc., where independent validation of the data was performed. Data validation procedures ultimately confirm each sample concentration to be either unqualified (i.e., identity and concentration of the constituent are certain) or qualified (i.e., the concentration, or possibly also the identity, of the constituent is not reliable). The various data qualifiers and the appropriate use of qualified data in risk assessment are addressed in USEPA guidance documents (USEPA 1989a, 1990a). Unqualified chemical concentrations were used in the risk assessment without modification. Qualified data were handled in the following manner:

- Data marked with a J-qualifier, indicating that the concentrations were estimated, were treated the same as unqualified data.

- For a given sample, substances that were also detected in various blank samples were not considered to be native unless the sample concentration exceeded by 5 times or more the level in the blank(s). For common laboratory contaminants (e.g., acetone, phthalate esters, methylene chloride, and toluene), the sample concentration had to exceed the concentration in the blank(s) by ten times or more to be considered native to the samples. Aqueous and solid sample results within 5 or 10 times the level in the blanks of a similar matrix (viz., aqueous or solid blank) were qualified as "not detected." Solid sample results within 5 or 10 times the level in aqueous field blanks were qualified as "qualitatively suspect."

- Qualified data marked with an R-qualifier, indicating an unreliable result, were eliminated from the data set.

B. Summary Statistics

In the RI report, 144 chemicals detected at least once in various sampled media were retained for further evaluation in the EA (see Section 4 of the RI). Table 1 identifies these chemicals and presents the environmental media in which they were detected. Summary statistics, including frequency of detection, minimum and maximum detected levels, and the range of reported quantitation limits for each chemical that was detected in various sampled media are presented in Tables 2 through 14 (viz., on-site test pit soil (Table 2), on-site pond borings (Table 3), ground water (Table 4), air (Table 5), on-site sediment (Table 6), on-site surface water (Table 7), off-site soil borings (Table 8), Crane-Deming soil (Table 9), test pit soils along railroad tracks (Table 10), MFLBC surface water (Table 11), MFLBC sediment (Table 12), MFLBC fish (Table 13), and MFLBC flood plain soil (Table 14). In developing these summary statistics, for duplicate or repeat samples, the highest of two or more reported concentrations, rather than their average concentration, was used for the purpose of estimating frequency of detection.

C. Reduction in the Number of Chemicals to Be Quantitatively Considered in the EA

Many of the 144 chemicals detected at least once are unlikely to contribute significantly to overall public health or environmental risk because of low frequency of detection, low detected concentrations, or comparatively low intrinsic toxicities compared with other chemicals detected at the Site. Consequently, the USEPA (1989a) permits a baseline risk assessment to be based upon a subset of all detected substances that is developed by considering three criteria: 1) the frequency of detection; 2) essential nutrient information; and 3) a concentration-toxicity screen. The chemicals eliminated based on these criteria are discussed below and summarized in Table 15.

1. Frequency of Detection

Based on guidance by USEPA (RAGS, p. 5-22), any chemical that was detected in less than five percent of the samples taken in each on-site medium is eliminated from further consideration in the risk assessment. A total of 31 chemicals were eliminated from further consideration in the risk assessment based on this criterion, leaving a total of 113 chemicals for further consideration.

2. Essential Nutrients

A number of trace elements that are present naturally in the environment are essential nutrients. A deficiency in these elements can result in impairment of biological functioning. In recognition of this, guidance for the conduct of risk assessments in RAGS (USEPA 1989a, p. 5-23) states that essential nutrients need not be considered in the quantitative public health risk assessment. Consistent with this guidance, the following eight essential nutrients were not considered further in the public health risk assessment for the Site: 1) calcium; 2) copper; 3) iron; 4) magnesium; 5) manganese; 6) potassium; 7) sodium; and 8) zinc. Thus, a total of 105 chemicals remain in consideration.

3. Concentration and Toxicity Screen

As stated in RAGS (p. 5-23), the objective of the concentration and toxicity "screening procedure is to identify chemicals in a particular medium that - based on concentration and toxicity - are most likely to contribute significantly to risks calculated for exposure scenarios involving that medium, so that the risk assessment is focused on the 'most significant' chemicals."

In this screening procedure, each chemical in a medium is scored according to its concentration and toxicity to obtain a risk factor as follows:

$$R_{ij} = (C_{ij})\ (T_{ij})$$

where

R_{ij} = risk factor for chemical i in medium j;

C_{ij} = maximum concentration of chemical i in medium j; and

T_{ij} = toxicity value for chemical i in medium j (either the cancer slope factor or the reciprocal of the reference dose, 1/RfD)

Slope factors and RfDs for the chemicals evaluated in the concentration and toxicity screen are presented in Appendix A. Additional discussion of slope factors and RfDs appears in Chapter IV, Toxicological Assessment.

Total chemical scores are calculated for each medium by summing all chemical-specific risk factors as follows:

$$R_j = R_{1j} + R_{2j} + R_{3j} + ... + R_{ij}$$

where

R_j = total risk factor for medium j; and

$R_{1j} + ... + R_{ij}$ = risk factors for chemicals 1 through i in medium j.

A separate R_j is calculated for carcinogenic and noncarcinogenic effects for each medium. As stated in RAGS (p. 5-24), "the ratio of the risk factor for each chemical to the total risk factor (i.e., R_{ij}/R_j) approximates the relative risk for each chemical in medium j." Therefore, chemicals whose R_{ij}/R_j ratios are very low compared with the ratios of other chemicals are eliminated from the risk assessment. As recommended in RAGS (p. 5-24), a ratio of 0.01 was used to eliminate chemicals from further consideration in the risk assessment (i.e., all chemicals in a medium whose R_{ij}/R_j ratios were less than 0.01 were eliminated from the risk assessment for that medium).

The calculations conducted for the concentration and toxicity screen are summarized in tables presented in Appendix B. Of the 105 remaining chemicals, a total of 19 chemicals could not be scored using the concentration and toxicity screen because no toxicity values were available. These chemicals were eliminated from the quantitative risk assessment, but are discussed qualitatively in the next chapter, Toxicological Assessment. Thus, 86 chemicals were scored using the concentration and toxicity screen.

Of these 86 chemicals, a total of 53 were eliminated from further consideration as a result of the screen. Therefore, 33 chemicals were retained for consideration in at least one environmental medium in the risk assessment. These chemicals are summarized in Table 16. Sixteen chemicals are retained in ground water; 3 in on-site sediment; 9 in on-site soil; 12 in on-site surface water; 12 in off-site soil; 4 in on-site air; 2 in the surface water of the MFLBC; 2 in fish from the MFLBC; 8 in the sediment of the MFLBC; and 2 in the flood plain soil.

Kepone was one of the chemicals eliminated from further consideration as a result of the concentration and toxicity screen. Kepone was detected infrequently at relatively

low concentrations in only a few on-site environmental media (viz., 6 out of 75 on-site test pit soil samples at a maximum detected concentration of approximately 85 ppb; 3 out of 63 on-site pond boring samples at a maximum detected concentration of approximately 761 ppb; 6 out of 145 ground water samples at a maximum detected concentration of approximately 13 ppb; and 1 out of 8 on-site surface water samples at a maximum concentration of approximately 0.3 ppb). Although Ruetgers-Nease reports that kepone was never produced at the Salem Site, its presence in these samples is not unexplainable, however, because kepone is one of the reported products of the environmental degradation of mirex (Carlson et al. 1976).

D. Consideration of Tentatively Identified Compounds (TICs)

Tentatively identified compounds (TICs) were reported for all media except flood plain soil, for which the only analytes quantified were mirex and photomirex. The list of TICs detected in each medium are reported in Appendix C. The number of TICs tentatively identified in each medium are summarized below.

Medium	Number of Reported TICs
On-site test pit Samples	40
On-site pond borings	49
Ground water samples	256
Air samples	13
On-site sediments	121
Off-site soil boring samples	17
Crane-Deming soil	10
Railroad track test pit samples	22
MFLBC surface water samples	4
MFLBC sediment samples	22
MFLBC fish tissue samples	49

A detailed discussion of TICs by medium and the likelihood that the TICs would significantly contribute to risk estimates developed in this assessment is provided in Appendix C. In any assessment of TICs, it is important to take into account that the assigned identity of a TIC is, in most cases, highly uncertain. Further, estimates of concentrations of TICs are highly uncertain and could be orders of magnitude higher or lower than the actual

concentration (see RAGS, p. 5-18). Recognizing these uncertainties in the available data for TICs, the following preliminary conclusions can be reached.

Samples of on-site soils, sediment, ground water, and air and railroad track test pits contain various halogenated organic compounds, PAHs and other aromatic compounds that are not likely to be components of natural biological systems. In all cases, analysis of target list compounds in the same medium show a similar spectrum of halogenated and aromatic compounds to be present. Therefore, a quantitative assessment of potential risks associated with these TICs, if one could be performed, would be unlikely to change the overall conclusions of potential risk associated with chemicals in Site media. Other compounds tentatively identified in on-site and railroad track test pit samples are relatively simple compounds (alkanes, alkenes) that would be expected to degrade rapidly. Some TICs in the sampled media, ground water in particular, were not identified sufficiently to allow conclusions to be made of potential risk.

The majority of TICs in off-site soil borings and soil samples from Crane-Deming are naturally-occurring compounds or, like benzeneacetic acid, are relatively simple compounds that would likely degrade rapidly in the environment. Only two halogenated hydrocarbons and three PAHs were tentatively identified in these off-site soils.

The vast majority of the TICs detected in fish, sediment, and surface water are natural components of biological systems and, as such, present no risk to potentially exposed populations. A few additional TICs are either present only in one or a few samples at low concentrations (e.g., benzo(e)pyrene) or are simple molecules that are likely to degrade readily (e.g., benzeneacetic acid) and, as such, are not likely to add significantly to risk. The remaining TICs are insufficiently identified to permit any assessment; however, most of these appear to be relatively simple molecules that would be expected to degrade rapidly.

E. Summary

In summary, 33 chemicals were retained for consideration in the quantitative risk assessment. These are:

<u>Volatile Compounds</u>

1,1,2-Trichloroethane
1,1,2,2-Tetrachloroethane
1,2-Dichloroethane
1,2-Dichloroethene (total)
Acetone
Benzene

Carbon tetrachloride
Chlorobenzene
Chloromethane
Tetrachloroethene
Trichloroethene
Vinyl chloride

Semivolatile Compounds
1,2-Dichlorobenzene
2,4-Dichlorophenol
4-Methylphenol
Benzo(a)anthracene
Benzo(a)pyrene
Benzo(b)fluoranthene
Benzo(k)fluoranthene
Bis(2-ethyl hexyl)phthalate
Hexachlorobenzene
Hexachlorobutadiene
Hexachloroethane
Indeno(1,2,3-cd)pyrene
n-Nitrosodiphenylamine
Pyrene

Pesticides
4,4'-DDT
Dieldrin

Other Organics
Mirex
Photomirex

Inorganics
Arsenic
Beryllium
Cadmium

IV. TOXICOLOGICAL ASSESSMENT

A. Introduction

To assess the potential health risks associated with exposure to chemicals evaluated quantitatively in the risk assessment, it is necessary to examine the relevant toxicologic literature to determine the effects in humans or laboratory animals of chemical exposure as a function of exposure levels. The USEPA has conducted such assessments on many frequently occurring environmental chemicals and has developed standardized toxicity values for use in risk assessment. These toxicity values -- reference doses (RfDs) and reference concentrations (RfCs) for noncarcinogenic chemicals and the noncarcinogenic effects of potential carcinogens, and cancer slope factors (SFs) and unit risks for known, suspected, or possible human carcinogens -- are published by the Agency and updated regularly (USEPA 1992a, USEPA 1993). It should be noted, however, that USEPA has not developed toxicity values for all chemicals evaluated in the risk assessment (e.g., photomirex, as discussed later in this chapter).

An RfD (or RfC) is USEPA's estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime. Unless adequate human data are available, an RfD is generally based on a study of the most sensitive animal species tested and is calculated based on the most sensitive endpoint measured. From this critical study, the experimental exposure representing the highest dose level tested at which no adverse effects were demonstrated (the no-observed-adverse-effect level, NOAEL) is identified. The RfD is derived from the NOAEL for the critical toxic effect by dividing the NOAEL by uncertainty (or safety) factors. These factors generally consist of multiples of 10, with each factor representing a specific area of uncertainty in the extrapolation from the available data. A 100-fold uncertainty factor is typically used to extrapolate results of long-term studies in experimental animals to humans, with additional factors applied where there are limitations in the available experimental data. Consequently, the RfD derived by this process does not provide a sharp demarcation between "safe" and "unsafe" levels of exposure. If the exposure level exceeds the RfD, there may be concern for noncancer effects. Because of the substantial safety factors incorporated in the RfD,

however, an exposure in excess of the RfD does not indicate that adverse effects will necessarily occur.

In assessing carcinogenic potential, USEPA uses a two-part evaluation in which the first step involves evaluating the likelihood that the substance is a human carcinogen (i.e., a weight-of-evidence assessment), and the second step involves defining the quantitative relationship between dose and response (i.e., development of a SF). In assessing the carcinogenic potential of a chemical, USEPA classifies a chemical into one of five groups based on the weight of evidence from human and animal investigation. These groups are as follows (USEPA 1989a, 1992a):

Group A: Human Carcinogen (sufficient evidence of carcinogenicity in humans)

Group B: Probable Human Carcinogen
B1 -- limited evidence of carcinogenicity in humans
B2 -- sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans

Group C: Possible Human Carcinogen (limited evidence of carcinogenicity in animals and inadequate or lack of human data)

Group D: Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)

Group E: Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies).

As noted above, the output of the second part of the evaluation is the derivation of a SF. A SF represents the upper 95 percent confidence limit on the linear component of the slope of the tumorigenic dose-response curve in the low-dose (low-risk) region. The cancer SF is derived by applying a mathematical model to extrapolate from the relatively high doses administered to experimental animals to the lower exposure levels expected for human contact in the environment. A number of low-dose extrapolation models have been developed. Each is based on general theories of carcinogenesis or certain statistical principles rather than on tumor data for the specific chemical of interest. USEPA generally uses the linearized multistage model in cancer risk assessment. Other models are available,

but generally predict lower cancer potency estimates than the linearized multistage model. The latter model does not necessarily provide the most "correct" or "accurate" measure of carcinogenic potency, but is used by USEPA in part as a policy matter to provide a conservative (i.e., health protective) estimate of potential carcinogenic potency.

B. Toxicity Values for Chemicals Evaluated in the Risk Assessment

Where available, USEPA-derived toxicity values have been used in this assessment. Chronic RfD and RfC values for the noncarcinogenic effects of chemicals and SFs and unit risks for carcinogens for all of the constituents evaluated in this assessment are summarized in Appendix A, along with the bases for these values. For a limited number of the chemicals detected at the Site, USEPA-derived toxicity values were not available. These included photomirex, kepone, and 19 additional chemicals. In addition, a SF was unavailable for mirex. In-depth, independent evaluations of the available toxicological data were conducted for mirex, photomirex, and kepone because of the particular significance of these three chemicals at the Site and because of the existence of toxicological data bases for these chemicals.

As stated above, USEPA-derived toxicity values, where available, have been used in this assessment; however, as pointed out in Chapter VIII in the discussion of uncertainties associated with the risk assessment process, differences of opinion exist among scientists with respect to some of the underlying assumptions made in estimating these values. Furthermore, the risks estimated using these toxicity values must be interpreted in light of the conservative assumptions built into the toxicity values. These underlying assumptions are also discussed further in Chapter VIII, Risk Characterization.

The remainder of this section provides a brief overview of the toxicity values for mirex, photomirex, and kepone derived for purposes of this assessment; more detailed documentation of the data used to derive these values is provided in appendices to this report.

1. Mirex

USEPA has developed a verified RfD for mirex of 2×10^{-4} mg/kg/day, based on liver and thyroid effects in a chronic study in the rat (NTP 1990). This RfD has been used in the current assessment.

Ruetgers-Nease has sponsored a reevaluation of the potential carcinogenicity of mirex. The Weinberg Consulting Group Inc. performed this evaluation for Ruetgers-Nease and recently submitted a petition to the Integrated Risk Information System

(IRIS) Information Submission Desk requesting that the USEPA reconsider the Weight-of-Evidence classification for mirex and its cancer slope factor. As presented in this petition, the currently available data on the potential carcinogenicity of mirex suggests that it should be classified in Weight-of-Evidence Group C, "possible human carcinogen," and that a slope factor of 0.34 $(mg/kg/day)^{-1}$ more accurately reflects its potential cancer potency. The petition to the IRIS Information Submission Desk on the carcinogenicity of mirex, which is the basis for the mirex carcinogenicity assessment, is contained in Appendix D.

USEPA has not yet completed its review of the cancer slope factor petition for mirex. However, the Agency has indicated that it ultimately may classify mirex in Weight-of-Evidence Group B2, "probable human carcinogen." In addition, the Agency has recommended that an interim cancer slope factor be calculated by modifying the slope factor proposed in the mirex cancer slope petition to reflect the use of a cross-species scaling factor based on the body surface areas of the test and target species (i.e., expressing mirex dosed in terms of daily amount administered per unit of body weight raised to the 2/3 power). This differs from the proposal in the mirex petition to use a cross-species scaling factor based on body weight raised to the 3/4 power, an approach recently proposed by the USEPA for used in cancer risk assessment (57 FR 24152, June 5, 1992). The cancer slope factor calculated using these recommendations of USEPA is 0.53 $(mg/kg/day)^{-1}$. This slope factor has been adopted in the current assessment. Because of the uncertainty in its ultimate classification, the Weight-of-Evidence classification for mirex will be B2/C.

2. Photomirex

The USEPA has not developed toxicity criteria for photomirex. A review of the toxicological data for photomirex was performed by the Weinberg Consulting Group Inc. to determine the suitability of the available data for calculating toxicological criteria (Appendix E). This information has been provided to Region V USEPA, the Ohio Department of Health, and the Agency for Toxic Substances and Disease Registry (ATSDR).

As described in the photomirex toxicity review (Appendix E), a chronic oral RfD for photomirex of 0.00125 mg/kg/day was derived, based on a reproductive toxicity study in the rat by Chu et al. (1981). Also, as described in the toxicity review, consideration of the available data on the potential carcinogenicity of photomirex indicates that this compound is most appropriately classified in Weight-of-Evidence

Group D, "not classifiable as to human carcinogenicity." This classification is generally used for agents with inadequate human and animal evidence of carcinogenicity or for which no data are available. For the purposes of this risk assessment, the Weinberg Group's recommendation concerning the toxicity criteria for photomirex has been adopted.

3. Kepone

The USEPA has not developed an RfD or SF for kepone. A review of the toxicological data for kepone was performed by the Weinberg Consulting Group Inc. to determine the suitability of the available data for calculating toxicological values (Appendix F). This information has been provided to Region III USEPA, and recommendations of toxicity criteria presented in the kepone toxicity review were accepted for use in a risk assessment included in a Region III Superfund Remedial Investigation.

As described in the kepone toxicity review (Appendix F), a chronic oral RfD for kepone of 6.5×10^{-4} mg/kg/day was derived, based on a 128-day mouse study by Good et al. (1965).

Three studies are available that provide primary testing data on which to evaluate the carcinogenic potential of kepone, specifically NCI (1976), Larson et al. (1979), and Sirica et al. (1989). As discussed in the toxicity review for kepone, these studies provide no evidence that kepone is carcinogenic in humans, and demonstrate that the animal data on carcinogenicity are equivocal. In light of these data, Weinberg recommended a designation of kepone as a Weight-of-Evidence Group C carcinogen ("possible human carcinogen"), and that a cancer slope factor for kepone not be developed due to inadequacies in the available data. Consistent with this recommendation, no evaluation of potential carcinogenic risk was conducted for kepone.

C. Chemicals for which No Toxicity Values Were Available

Toxicity values (i.e, RfDs and RfCs for noncarcinogenic effects and SFs or unit risks for carcinogens) were not available for 19 chemicals or chemical classes detected in Site media. Therefore, these chemicals were not included in the concentration and toxicity screen that was used to select chemicals to be evaluated quantitatively in the risk assessment. The chemicals and chemical classes for which toxicity values were not available are listed below.

Semivolatile Compounds

- Acenaphthylene
- Dibenzofuran
- 1,3-Dichlorobenzene
- Diphenyl sulfone
- 2-Methylnaphthalene
- 2-Nitrophenol
- Phenanthrene

Dioxins and Furans

- Total HpCDDs
- Total HpCDFs
- Total HxCDDs
- Total HxCDFs
- Total PeCDDs
- Total PeCDFs
- Total TCDD
- Total TCDFs

Inorganic Compounds

- Aluminum
- Cobalt
- Lead
- Thallium

With the exception of certain polychlorinated dibenzo-p-dioxin and furan (PCDD and PCDF) congeners, none of the 19 chemicals are considered by USEPA to be potential human carcinogens. Because of the carcinogenic potential of certain dioxin and furan isomers, further consideration is given to the significance of the analytical results for total PCDDs and PCDFs below. Of the 19 chemicals, diphenyl sulfone is noteworthy because of its relatively high frequencies of detection in several site media (e.g., on-site test pits, 32/80; ground water, 52/144). Additional consideration of the toxicity of this compound is therefore presented below. Finally, an RfD is not available for lead because the potential noncarcinogenic effects of lead are evaluated using an alternative methodology (the USEPA Uptake Biokinetic (UBK) model) to the RfD methodology used for other chemicals. This

methodology and the potential for lead to significantly contribute to total Site risk is discussed further in section C.3.

1. Total Polychlorinated Dioxins and Furans

Analytical results for Site media included data for individual PCDD and PCDF congeners, as well as total PCDDs and PCDFs.

USEPA has concluded that cancer risk may be associated with exposure to some, but not all, PCDDs and PCDFs. This conclusion is based on the results of long-term cancer studies in animals with a few of the individual PCDD congeners, or mixtures of PCDD congeners (specifically 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) and a mixture of 1,2,3,6,7,8-hexachlorodibenzo-p-dioxin and 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin), plus short-term experimental studies indicating similarities in the mechanism of toxicity of various PCDD and PCDF congeners (USEPA 1987a, 1989c).

The various PCDD and PCDF congeners differ in their degree of toxicity (CDTSC 1992). To address this issue, USEPA has developed a procedure for estimating the toxicologically equivalent concentration of 2,3,7,8-TCDD, based on the relative toxicity of the different congeners to 2,3,7,8-TCDD in certain short-term tests (USEPA 1989c). These "toxicity equivalence factors" (TEFs) are used to estimate the toxicologically equivalent concentration (TEQ) of 2,3,7,8-TCDD in various media. The TEFs recommended by USEPA are for congeners with chlorines in the 2,3,7,8- positions only. In the concentration and toxicity screen and in the quantitative risk assessment, TEFs were used, as appropriate, for individual PCDD and PCDF congeners detected in Site media. The TEF approach was not applied to total concentrations of isomers and congeners. To the extent that quantifiable concentrations of congeners with chlorines in the 2,3,7,8- positions contributed to total PCDDs and PCDFs, these congeners were included in the risk assessment.

2. Diphenyl Sulfone

Diphenyl sulfone was detected in on-site soils and sediments, ground water, and off-site surface water and sediment relatively frequently at relatively elevated concentrations (e.g., maximum detected concentrations in on-site soil and ground water of 7,400 mg/kg and 14 mg/L, respectively).

The available toxicity data for diphenyl sulfone is limited to an LD_{50} of 320 mg/kg in the rat (NIOSH 1990). An LD_{50} is the single dose calculated to be lethal to 50 percent of the animals, and is generally not considered a sufficient basis for evaluating

the toxicity of a chemical associated with chronic low-dose exposures. Although acute toxicity is not necessarily a predictor of chronic toxicity, it is worth noting that the LD_{50} for diphenyl sulfone is substantially higher than the LD_{50} for other chemicals included in the quantitative risk assessment. Although the lack of adequate toxicity data for diphenyl sulfone to permit quantitative assessment of risk introduces some uncertainty in the risk assessment, it is unlikely that diphenyl sulfone would significantly add to the overall risk of those chemicals evaluated quantitatively in this assessment.

3. Lead

The potential risk from exposure to lead is typically evaluated using USEPA's computerized Uptake Biokinetic (UBK) model, LEAD 0.6, which predicts blood lead concentrations in populations of children (ages 0 to 7) exposed to lead through a variety of media. The model is designed to estimate blood lead levels using a combination of default exposure assumptions and geometric mean exposure concentrations combined with site-specific exposure information where applicable.

Lead was detected in on-site test pit soil, on-site soil (pond), off-site soil borings, and ground water. The maximum concentrations in both on- and off-site soil (i.e., 26.9 ppm and 0.2 ppm, respectively) were significantly lower than the default soil concentration recommended by USEPA for use in the model (i.e., 200 ppm). The geometric mean ground water concentration (i.e., 13 mg/L) was higher than the recommended default value in the model. Therefore, the UBK model was run using the geometric mean ground water concentration in combination with the default soil concentration and default values in the model for contributions to lead intake from air, dust, and paint. This model run indicated that the geometric mean blood lead levels associated with such an exposure would not exceed the level generally considered acceptable by USEPA (i.e., a blood lead level of 10 ug/dL or less in 95 percent of exposed children).

Based on the qualitative analysis of chemicals excluded from the quantitative risk assessment for lack of toxicity values (with a focus on dioxans and furans, diphenyl sulfone, and lead for the reasons discussed above), it can be concluded that the 19 chemicals and chemical classes for which no toxicity values were available are unlikely to contribute significantly to risk associated with the Site.

V. IDENTIFICATION OF EXPOSURE PATHWAYS

A. Introduction

In this chapter, potential exposure pathways under current and hypothetical future land-use conditions of the study area (i.e., Site, adjacent areas, and locations along the MFLBC) are identified. Exposure pathways are those situations by which a population or an individual could be exposed to chemicals present in the study area. The identification of potentially exposed populations and potential exposure pathways is based primarily on information presented in the RI report (ERM 1993), site-specific information obtained from ENVIRON's visits to the study area, knowledge of existing chemical concentrations in the study area, local land-use patterns, activities of nearby residents, and judgments about what constitutes reasonable behavior.

B. Characterization of Exposure Scenarios

For purposes of this baseline risk assessment, potential exposures under both current and hypothetical future land uses of the study area are evaluated. A current exposure scenario evaluates whether there is a potential health threat under existing land use conditions. A future exposure scenario evaluates whether there is a potential health threat under hypothetical future land use conditions (but assuming current environmental concentrations).

The following populations have been identified as having the potential to be exposed to chemicals present in the study area under current or future exposure scenarios. Populations are identified separately for the Site, areas adjacent to the Site, and locations along the MFLBC. The populations identified for locations along the MFLBC are the same as those identified in the previous draft of the EA (ENVIRON 1991).

1. Site

- Trespassers to the Site are included in both the current and future exposure scenarios.

- Although the future development of the Site for industrial use is remote, in accordance with USEPA guidance (1991a), an on-site worker is included in the future exposure scenario.

- Although the future residential development of the Site is remote, in accordance with USEPA guidance (1991a), an on-site resident is included in the future exposure scenario.

2. Areas Adjacent to the Site

- Off-site workers in areas adjacent to the Site are included in both the current and future exposure scenarios.

- Off-site residents in areas adjacent to the Site are included in both the current and future exposure scenarios.

3. Locations Along the MFLBC

- Recreational visitors, who are assumed to engage in activities (such as fishing and hunting) in and along the MFLBC, are included in both the current and future exposure scenarios.

- Off-site residents, whose properties are located within the flood plain of the MFLBC, are included in both the current and future exposure scenarios.

C. Identification of Exposure Pathways

Potential exposure pathways identified by ENVIRON for the study area are primarily based upon those recommended for evaluation by USEPA (1991a). The potential exposure pathways identified for locations along the MFLBC are also based on information obtained from a survey conducted by ODH in September 1989 (see Appendix J). Data on potential exposure to mirex among persons living in the vicinity of the MFLBC were obtained through questions concerning fishing and recreational contact with the MFLBC, consumption of game and farm products, and employment history. The ODH survey methodology was designed to identify people with the greatest likelihood of exposure. Specifically, 575 questionnaires were mailed to residents in the vicinity of the MFLBC between Salem and Lisbon, and more

specifically to residents who lived on roads that ran adjacent to the creek. An additional 100 questionnaires were placed in public libraries in Salem and Lisbon; other attempts were made to recruit subjects through local newspaper announcements and at public meetings.

The pathways identified by ENVIRON are described below and are summarized in Table 17.

- Ingestion of ground water. Based on information presented in the RI report (ERM 1993), there are no known current exposures to chemicals present in the ground water in the study area. Although the possibility is remote, on-site ground water could be used by on-site workers or on-site residents as a source of drinking water in the future. Potential exposure to these receptors is quantitatively assessed for the ingestion of ground water pathway. Potential exposure via dermal contact with ground water and via inhalation of vapors from ground water during showering or other uses are qualitatively assessed. Results from the quantitative assessment of on-site ground water are used to qualitatively evaluate the potential future exposure to ground water by an off-site resident and off-site worker.

- Ingestion of soil. During activities such as working, playing, or gardening, various populations in the study area may be exposed to chemicals present in soils. Potential exposure via incidental ingestion of soil is quantitatively assessed for trespassers on the site, residents (on-site, at areas adjacent to the Site, and at locations along the MFLBC), and workers (on-site and at areas adjacent to the Site). Results from the quantitative assessment of the ingestion of soil pathway are used to qualitatively evaluate potential exposure via dermal contact with soils.

- Inhalation of air. Potential exposure to chemicals detected in on-site air (which is likely primarily due to volatilization from on-site soils) is quantitatively assessed for the potential on-site receptors (trespasser, resident, and worker). Results from the quantitative assessment of this pathway are used to qualitatively evaluate the potential exposure to chemicals present in air for receptors in adjacent off-site areas. In addition to chemicals volatilizing to the air from soils, chemicals may also volatilize from ground water that discharges to the surface in the form of seeps. Seeps are known to have occurred along the railroad tracks and on Crane-Deming property and have the potential to exist at other locations. The elimination of these ground water seeps is an Interim Remedial Measure goal for

which Ruetgers-Nease is negotiating an Administrative Order by Consent. Based on the above, the volatilization of chemicals from ground water seeps is not considered in the risk assessment.

- Ingestion of surface water and sediment. During activities such as fishing, swimming, and wading, potential exposure to chemicals present in the surface waters or sediments of the MFLBC may occur. Potential exposure is quantitatively assessed for the incidental ingestion of surface water and sediment pathways. Populations potentially exposed via these pathways are assumed to be recreational visitors along the MFLBC. Because an advisory against fishing, wading, and swimming is in effect, current exposures to the section of the MFLBC within the area of advisory is likely to differ from portions of the creek outside the advisory area. Results from the quantitative assessment of the ingestion of surface water and sediment pathways are used to qualitatively evaluate potential exposure via dermal contact with surface water and sediment. In addition to the above, potential exposure to chemicals present in on-site surface water and sediment is quantitatively assessed for the on-site trespasser.

- Ingestion of fish. Potential exposure to chemicals present in fish in the MFLBC may occur via the ingestion of fish. As above, current exposures are likely to differ for sections of the MFLBC within and outside the advisory area. The population considered to have the greatest potential for exposure via this pathway is the recreational visitor.

- Ingestion of game. Potential exposure to chemicals may occur via the ingestion of game hunted or trapped in the area along the MFLBC. The population considered to have the greatest potential for exposure via this pathway is the recreational visitor.

- Ingestion of vegetables. Potential exposure to chemicals present in soils may occur via the consumption of vegetables that accumulate chemicals from these soils. This pathway is quantitatively assessed for residents (on-site, at areas adjacent to the Site, and at locations along the MFLBC).

- Ingestion of beef and milk. Because cattle with access to the MFLBC may accumulate chemicals present in the flood plain soil, the ingestion of beef or milk by residents (farmers and their families) is quantitatively assessed. This potential exposure is included as a future exposure scenario. Potential exposures of farmers are not characterized under the current exposure scenario because fences and bridges on farms adjacent to the MFLBC prevent access of livestock to the creek and flood plain. Potential exposures to chemicals in beef and milk are characterized under the future exposure scenario because USEPA guidance (1989a) requires these exposures to be evaluated as if these remedial measures were not put in place (i.e., in the absence of any actions to control or mitigate releases).

VI. ESTIMATION OF ENVIRONMENTAL CONCENTRATIONS

A. Introduction

To assess the potential risks within the study area, estimates of chemical concentrations in the following environmental media are necessary:

- ground water
- surface soil
- outdoor air
- surface water
- sediment
- fish
- game
- vegetables
- beef and milk

Estimates of chemical concentrations for all environmental media, except vegetables, are based on sampling data. Estimates of chemical concentrations in vegetables are based on a mathematical model that relates vegetable concentrations to soil concentrations.

As discussed in Chapter VII, Estimation of Human Intake, reasonable maximum exposure (RME) scenarios are evaluated as part of the baseline risk assessment. In accordance with USEPA guidance (1989a), the RME concentration for a chemical is represented by either the highest observed (detected) concentration or the 95 percent upper confidence limit on the mean concentration (95% UCL), whichever is lower. Recent USEPA guidance (1992b) states "EPA's experience shows that most large or 'complete' environmental contamination data sets are lognormally distributed rather than normally distributed." While USEPA guidance provides methods for generating the 95 percent UCL that are specific for both the normal and lognormal distributions, ENVIRON generally and conservatively assumed that all data sets were lognormally distributed.

For lognormal distributions, the 95 percent UCL was calculated using the H-statistic developed by Land, which was described in recent USEPA guidance (USEPA 1992b).[3] In a few cases, the calculated 95 percent UCL value was found to be lower than the arithmetic mean. In such cases, the 95 percent UCL was recalculated assuming a normal distribution.

For those substances where a non-detect value was reported for a given sample, it was assumed that the actual sample concentration was one-half of the sample quantitation limit, which was determined to be the product of the quantitation limit multiplier and the quantitation limit.

B. Ground Water

The RME concentrations for chemicals evaluated in ground water are presented in Table 18. These concentrations are used in modeling exposures to hypothetical future on-site residents and on-site workers. In accordance with USEPA Region V guidance (USEPA 1991b), ENVIRON calculated the RME ground water concentration based on three monitoring wells located in the center of the upper bedrock aquifer's plume. This aquifer was selected because it is the only aquifer with sufficient yield to potentially support a drinking water well in the future.

Upon review of the data, ENVIRON and ERM determined that the three wells with the highest concentrations in the upper bedrock aquifer are RNS-GW-D12, RNS-GW-T2, and RNS-GW-RW1. Because of the limited number of samples taken at these three wells (six total samples in two rounds of sampling), ENVIRON elected not to conduct a statistical analysis of the data. Rather, ENVIRON conservatively assumed, per USEPA Region V guidance (USEPA 1991b), that the maximum detected concentration from both rounds of sampling for these three wells represented the RME value.

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[3] Because the number of samples taken within a specific exposure study area is generally limited, a particular data set could theoretically be statistically evaluated as being both normally and lognormally distributed. Because calculation of the 95% UCL for lognormal distributions using the H-statistic typically provides a more conservative estimate of the RME concentration than the Student-t statistic, the data were assumed to be lognormally distributed. The H-statistic gives an exact 95% UCL for the population mean only if the underlying distribution is lognormal. It should be noted that in order to accurately obtain the H-statistic used in the Land equation, a cubic interpolation (four-point Lagrangian interpolation) is required. Because the number of data points is generally small, a linear interpolation was assumed to provide a reasonable approximation of the H-statistic.

C. Surface Soil

The RME concentrations for chemicals evaluated in surface soils[4] (i.e., on-site soil, adjacent site soil, and flood plain soils along the MFLBC) are presented in Tables 19 through 23. On-site surface soil concentrations outside the fenceline, used in modeling exposure to a current on-site trespasser, are presented in Table 19. It is assumed that a current trespasser would not climb the fence that surrounds a large portion of the Site. Concentrations in on-site surficial soil borings and test pits, both inside and outside the current fenceline, which were used in modeling exposures to hypothetical future on-site residents, hypothetical future on-site workers, and hypothetical future trespassers, are presented in Table 20. In these hypothetical future on-site use scenarios, it is assumed that the existing fence is no longer in place. Off-site concentrations in surface soils on the Crane-Deming property, used in modeling exposure to current and hypothetical future workers on properties near to the Site, are presented in Table 21. Concentrations in off-site soil borings, used in modeling exposure of current and hypothetical future residents on properties adjacent to the site, are presented in Table 22. Finally, concentrations in flood plain soils, used in modeling exposure of current and hypothetical future residents living adjacent to the MFLBC, are presented in Table 23.

D. Outdoor Air

The RME concentrations for chemicals evaluated in ambient outdoor air in the vicinity of the Site are presented in Table 24. There were six air stations located in the vicinity of the Site. Based on data provided to ENVIRON, one or two air samples were collected at each of these stations. Because of the limited number of samples taken at these six monitoring stations, ENVIRON elected not to conduct a statistical analysis of the data. Rather, for each chemical considered ENVIRON conservatively assumed, similar to ground water, that the maximum detected air concentration from the six stations represented the RME value. Maximum detected concentrations for the various chemicals evaluated were relatively evenly distributed among the six monitoring stations.

E. Surface Water

The RME concentrations for chemicals evaluated in surface water are presented in Tables 25 and 26. On-site surface water concentrations, used in modeling exposures of a

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[4] In the RI, soil samples were taken at various depths. In the EA, surface soils were represented by the uppermost sampling interval. For samples taken from a test pit, the uppermost interval was typically zero to 6 inches. For samples taken from a soil boring, the uppermost interval was typically zero to three feet.

current and hypothetical future on-site trespasser, are presented in Table 25. Surface water concentrations along the MFLBC, used in modeling exposures of a current and hypothetical future recreational visitor, are presented in Table 26. Because of the low concentrations and small number of data points for MFLBC surface water samples, the maximum detected concentrations were used instead of the calculated RME values. In this instance, the calculated RME concentrations were equal to or slightly lower than their corresponding maximum detected concentrations.

F. Sediments

The RME concentrations for chemicals evaluated in sediments are presented in Tables 27 through 29. On-site sediment concentrations, used in modeling exposures of a current and hypothetical future on-site trespasser, are presented in Table 27. Sediment concentrations along the upstream reaches of the MFLBC (above Lisbon Dam), used in modeling exposures of a current and hypothetical future recreational visitor, are presented in Table 28. Sediment concentrations along the downstream reaches of the MFLBC (below Lisbon Dam), used in modeling exposures of a current and hypothetical future recreational visitor, are presented in Table 29.

G. Fish

The RME concentrations for chemicals evaluated in fish caught in the MFLBC above and below the Lisbon Dam are presented in Table 30. These concentrations are used in modeling exposures of a current and hypothetical future recreational visitor.

H. Vegetables

Chemical uptake by produce grown in home gardens is related to the location of gardens of potentially exposed populations (i.e., on-site, areas adjacent to the site, or along the MFLBC). For this assessment, chemical uptake by three classes of homegrown produce was evaluated. These three classes are:

- leafy aboveground produce (e.g., cabbage and lettuce);
- underground produce (e.g., carrots and potatoes); and
- non-leafy aboveground produce (e.g., tomatoes and cucumbers).

The uptake of chemicals by produce is a function of the chemical concentration in soil, physicochemical properties of the particular chemicals, and the relative affinity for chemical

uptake by the different classes of produce. The individual chemical concentrations used in assessing human exposure to vegetables are based on a weighted average for the three classes of produce, calculated as the relative percentages of each type typically ingested by consumers. A detailed development of the methodology used to estimate chemical concentrations in produce is presented in Appendix G[5]. Based on a review of the various chemicals found in soils in the study area, mirex and photomirex are most likely to bioaccumulate in plant or animal tissue because they are highly hydrophobic compounds. Because the other chemicals considered do not tend to bioaccumulate into organic material to the same extent as mirex and photomirex, their contribution to overall plant or animal uptake was assumed to be negligible. Therefore, bioaccumulation of all other chemicals was not evaluated.

The estimated concentrations of the chemicals (i.e., mirex and photomirex) in the three classes of produce are presented in Table 31. For hypothetical future on-site residents, chemical concentrations in homegrown produce are based on their RME concentrations in on-site surficial soil borings and test pits, both inside and outside the current fenceline. For current and hypothetical future off-site residents on property adjacent to the Site, chemical concentrations in homegrown produce are based on their RME concentrations in off-site soil borings. Finally, for current and hypothetical future residents living along the MFLBC, chemical concentrations in homegrown produce are based on their RME concentrations in flood plain soils.

I. Beef and Milk

Future residents along the MFLBC could be indirectly exposed to mirex and photomirex present in flood plain soils via the ingestion of locally-grown beef or dairy products. Cattle raised on property along the MFLBC could be exposed to mirex and photomirex through ingestion of soils and pasture grasses containing these chemicals. The estimated concentrations of mirex and photomirex in beef tissue and whole milk are presented in Tables 32 and 33, respectively. For both beef tissue and whole milk, the estimated RME concentrations are based on sampling data collected on local cattle. Measured data in beef

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[5] In the methodology presented in Appendix G, the estimated chemical concentration in produce is based on a unit chemical concentration in soil (i.e., 1 mg chemical/kg soil). Because chemical uptake by produce is linearly proportional to the chemical concentration in soil, the relative chemical concentration in produce is simply the RME concentration in soil multiplied by the calculated unit concentration based factor of 0.00093 mg/kg.

and milk were used to estimate exposure instead of modeled concentrations because of the large degree of uncertainty in bioaccumulation models used to estimate uptake in cows.

Data on mirex concentrations in locally-grown beef and milk were collected by the OEPA. Fourteen beef fat samples were collected in 1989 by the Ohio Department of Agriculture (ODA) from cows raised on one farm adjacent to the MFLBC. The 1989 beef fat samples had an average mirex concentration of approximately 0.22 mg/kg. This beef fat concentration is equivalent to a fresh meat mirex concentration of approximately 0.055 ppm, assuming a total body fat content of 25 percent for a cow. The actual fat content of ingested beef products could be less than 25 percent, but would unlikely be higher than this value. These 1989 results conflict somewhat with the results of several beef fat analyses collected in 1990 from cows raised on different farms along the MFLBC, which indicated lower mirex concentrations in beef fat than the 1989 sampling.

The RME concentration for beef fat was calculated to be 1.75 mg/kg. This corresponds to a fresh meat concentration of 0.44 mg/kg. This RME concentration is equal to the maximum detected concentration (assuming that the detection limit is equal to 0.01 mg/kg), where values reported as "trace" equal this assumed detection limit and non-detects equal one-half this detection limit. Because the maximum detected concentration was six times higher than the next highest sample, the standard deviation on the lognormalized data set was relatively large. This results in a calculated RME concentration that exceeds the maximum detected concentration. It can be demonstrated, however, that if the sample detection limit is assumed to be equal to the lowest detected concentration of 0.05 mg/kg (and assuming non-detects equal 0.025 mg/kg), the RME concentration would be 0.78 mg/kg, an approximately 50 percent reduction. Nevertheless, because no detection limit data were reported, ENVIRON chose the more conservative approach.

Similar to the beef fat samples, twelve milk fat samples collected from one local farm in March 1989 had an average mirex concentration of approximately 0.05 ppm. This corresponds to a whole milk concentration of mirex of approximately 0.002 ppm (assuming 3.68% fat in milk). The RME concentration for milk fat was calculated to be 0.13 mg/kg. This corresponds to a fresh milk concentration of approximately 0.005 mg/kg.

Because there is no sampling data or uptake information available for photomirex, the estimated RME concentration for photomirex in beef and milk is assumed to be linearly proportional to mirex uptake. As such the RME concentration of photomirex is equal to the mirex RME concentration multiplied by the ratio of photomirex to mirex in flood plain soils.

J. Game

Residue data for mirex in wildlife samples collected by the ODH were used for estimating concentrations of mirex in game. The ODH took samples of blood and fat from raccoon and opossums at nine sites along the MFLBC in September and October 1989. Trapping stations were located along the length of the creek from near the Site down to Beaver Creek State Park. Raccoon and opossum were chosen for this survey because the diet of these animals, which consists of a wide variety of plants and animals including fish, crayfish, and other aquatic animals, would provide the greatest potential for bioaccumulation of mirex.

The results of the ODH wildlife sampling (ODH 1990a) are included in Appendix H. Mirex levels in fat samples from the 22 animals trapped near the MFLBC ranged from non-detect (in 8 samples) to a maximum of 0.0527 ppm; levels in blood (serum) samples ranged from non-detect to 0.0089 ppm. The detection limit for the mirex analyses was not provided. In the absence of a reported detection limit, the average mirex concentration of 0.0153 ppm in fat obtained from raccoon and opossum was calculated by averaging only the samples with detectable mirex concentrations.

Because mirex readily partitions into fat, the concentration in the edible portion (i.e., fresh meat) of game was estimated from residue data in fat, based on the data for raccoons and beef cattle. The percentage of fat in raccoon meat (cooked or roasted) is approximately 14.5 percent (USDA 1975) and the percentage of separable fat (estimated from data for various cuts of beef) is about 75 percent (USDA 1975). No data are available on the percent fat content of opossum tissues; however, percentage of fat can reasonably be expected to be similar in raccoon and opossum tissue. Accordingly, the average mirex level in raccoon and opossum fat of 0.0153 ppm can be adjusted by the ratio 0.145/0.75 to give an estimated concentration in the edible portion of game of 0.003 ppm.

Other game, such as deer, would likely have substantially lower mirex residue levels because diets do not include fish or other food with high bioaccumulative potential and because of the lower fat content in the edible portion of deer meat (i.e., the fat content of deer meat is reported to be 4 percent; USDA 1975). Therefore, the concentration derived for game of 0.003 ppm is considered highly conservative for most hunters.

It should be noted that the ODH reviewed the wildlife residue data and concluded that "[t]here are no federal or state regulations for allowable concentrations of mirex in sport hunted or trapped (noncommercial) wild game, however, the Federal Food and Drug Administration tolerance level for mirex in commercial meat is 100 ppb. Mirex levels in ODH's study did not approach this level.... We do not believe that consumption of raccoons

and opossums hunted or trapped in the MFLBC watershed poses a significant risk to human health." (See Appendix H).

VII. ESTIMATION OF HUMAN INTAKE

A. Introduction

The next step in the risk assessment process is the estimation of the human intake received through exposure to the chemicals evaluated in the various environmental media. Chemical intakes (also referred to as Chronic Daily Intakes or CDIs) are expressed in terms of the mass of substance in contact with the body per unit body weight per time (or mg/kg/day), and are calculated as a function of chemical concentration in the medium, contact rate, exposure frequency and duration, body weight, and averaging time. The values for some of these variables are dependent upon conditions specific to the site and characteristics of the potentially exposed populations.

It is not possible to estimate accurately the exposures for potentially exposed populations due to uncertainties in both current and future behavior patterns of these populations, and due to limitations in knowledge of other exposure variable values. In light of this uncertainty, USEPA (1989a) recommends that intakes reflect an estimate of the reasonable maximum exposure (RME), defined as the highest exposure that is reasonably expected to occur. USEPA's intent with the RME "is to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures" (USEPA 1989a, p. 6-5). As discussed in the Exposure Factors Handbook (USEPA 1990b), USEPA recommends that not all values be at their individual maximum in calculating the RME; professional judgment can be used to combine values to arrive at a set of variables that adequately estimates the RME. Consistent with USEPA guidance (USEPA 1989a, 1990b), the estimates of human intake calculated in this risk assessment are those for RMEs.

In an exposure assessment, it is generally necessary to provide at least two different estimates of the CDI, one for noncarcinogenic effects and a second for carcinogens. The CDI generally used in the assessment of noncarcinogenic effects is the average daily dose an individual is likely to receive on any day during the period of exposure. In cases where exposure is intermittent, USEPA guidance states that it is appropriate to average the intake over the period of exposure. For potential carcinogens, the CDI is estimated by averaging

the total cumulative intake over a lifetime (USEPA 1989a).[6] This distinction in the calculation of the CDI for potential carcinogens and noncarcinogens relates to the currently-held scientific opinion that the mechanisms of action of the two categories of chemicals are different. For carcinogens, the assumption is made that a high dose received over a short period of time produces a carcinogenic effect comparable to a corresponding low dose spread over a lifetime (USEPA 1989a). It should be noted, however, that new information about the potential mechanisms of carcinogenesis suggests that such an assessment is not always warranted.

As previously described, estimates of human intake have been developed for populations potentially exposed under current or future land use conditions to on-site media, to areas adjacent to the Site, or to locations along the MFLBC. The populations are:

On-Site

- Trespasser (current and future land use)
- Worker (future land use)
- Resident (future land use)

Areas Adjacent to Site

- Worker (current and future land use)
- Resident (current and future land use)

Locations along the MFLBC

- Recreational visitor (current and future land use)
- Resident (current and future land use)

The specific assumptions used to estimate potential exposures of each of the potentially exposed populations are presented in Appendix I. A more general discussion of the assumptions used to estimate intakes for these populations is presented below. For the worker (as well as for the resident), several of the exposure assumptions for the on-site and

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[6] Averaging time (AT) for noncarcinogens and potential carcinogens will differ as follows: For noncarcinogens, the AT is the period over which exposure is assumed to occur (i.e., exposure duration (ED) x 365 days/year). For potential carcinogens, intakes are calculated by prorating the total cumulative dose over a lifetime (70 years). Therefore, the AT equals 70 years x 365 days/year or 25550 days.

off-site populations are common to both. Therefore, single discussions of worker and residential assumptions are provided for each in the following sections.

B. Discussion of Intake Assumptions for the Potentially Exposed Populations

1. On-site Trespasser

As discussed in Chapter V and summarized in Table 17, potential exposures of a trespasser onto the Site under current and future land use conditions have been evaluated quantitatively for the following potential exposure pathways:

- Ingestion of soil
- Inhalation of air
- Ingestion of surface water
- Ingestion of sediments

The equations and exposure assumptions used to estimate chemical intakes for the trespasser are presented in Appendix I. In general, the intake assumptions were constructed under the assumption that the types of populations most likely to trespass on Ruetgers-Nease property are children and teenagers. Therefore, for estimating exposures for the trespasser, the potentially exposed population was assumed to be school-age children exposed over a nine-year period as older children and young teenagers. Estimates of intake have been specifically developed using the physiologic parameters for a 12-year old as representative of this age group.

A trespasser is assumed to trespass onto Ruetgers-Nease property approximately 2 times per week during the summer months, or 24 days/year, and spend approximately 4 hours on-site during each trespassing event. Currently, some portions of the Site are fenced and any access to the fenced portions of the Site is considered unlikely. Therefore, current exposures for the on-site trespasser are based on soil concentrations in the unfenced portions of the Site. In the future, however, because the integrity of the fence cannot be assured, the on-site trespasser is assumed to be exposed to all on-site soils.

2. Intake Assumptions for the Worker

Intake estimates have been developed for a future on-site worker and for a worker employed on property adjacent to the Site under current and future land use conditions.

Potential exposures of a future on-site worker have been evaluated quantitatively for the following potential exposure pathways:

- Ingestion of ground water
- Ingestion of soil
- Inhalation of air

For the off-site worker, estimates of exposure have been evaluated quantitatively for exposure via incidental ingestion of soil. Potential current or future exposures via inhalation of chemicals in air or via ground water have been evaluated qualitatively (see Chapter VII, Risk Characterization).

In general, intake estimates have been modeled for a worker such as a grounds keeper who would be engaged in activities involving routine contact with outdoor media. The worker is assumed to be employed for 25 years, and to be exposed for 8 hours/day, 250 days/year, consistent with RAGS guidance (USEPA 1991a).

The specific equations and exposure assumptions used to estimate chemical intakes for the worker are presented in Appendix I.

3. Intake Assumptions for the Resident

Intake estimates have been developed for a resident exposed to on-site media under future land use conditions of the Site, for a resident whose property is adjacent to the Site and who could be exposed directly or indirectly to chemicals in off-site soil, and for a resident whose property extends along the MFLBC and who could be exposed directly or indirectly to chemicals in flood plain soil.

As discussed in Chapter V and summarized in Table 17, potential exposures of future on-site residents have been evaluated quantitatively for the following potential exposure pathways:

- Ingestion of ground water

- Ingestion of soil
- Inhalation of air
- Ingestion of homegrown vegetables
- Ingestion of beef and milk

Exposure pathways evaluated quantitatively for off-site residents (on property adjacent to the Site and along the MFLBC) are incidental ingestion of soil and ingestion of homegrown vegetables. Any current or future potential exposures of the residential population adjacent to the Site to chemicals in air or ground water have been evaluated qualitatively (see Chapter VIII, Risk Characterization). Several farms are located on property along the MFLBC. Although there is no current access of livestock to the creek, the assumption was made that in the future dairy and beef cattle raised on farms along the MFLBC could graze on the flood plain. Therefore, for the residential population along the MFLBC alone, potential future exposures to chemicals via ingestion of homegrown beef and milk were evaluated.

In evaluating intakes of all of the residential populations, exposure were assumed to occur 350 days per year for 30 years, consistent with guidance in RAGS (USEPA 1989a, 1991a).

With two exceptions, exposures for the resident have been modeled for each potential exposure pathway using parameters for the average adult. Available data suggest that the rate of ingestion of milk and incidental ingestion of soil during childhood are substantially greater on a mg/kg/day basis than are adult exposures. For these pathways, therefore, the RME exposure for a 1 to 6 year-old as well as an adult were modeled. In estimating exposures for these pathways, the RME exposure duration (ED) of 30 years was divided among the two age groups as follows: the ED for ages 1 to 6 was assumed to be 6 years and the ED for the adult was assumed to be 24 years.

The equations and exposure assumptions used to estimate intakes of chemicals for the resident are presented in Appendix I.

4. Intake Assumptions for a Recreational Visitor

As discussed in Chapter V and summarized in Table 17, potential exposure of a recreational population along the MFLBC has been evaluated quantitatively for the following pathways:

- Ingestion of sediment
- Ingestion of surface water
- Ingestion of fish
- Ingestion of game

The specific equations and assumptions used to estimate chemical intakes for the current and future recreational population along the MFLBC are presented in Appendix I.

With the exception of the pathway of incidental ingestion of sediments from the MFLBC, exposures for a recreational population have been modeled using parameters for an average adult population. As discussed for the residential populations, potential exposures via sediment ingestion (using soil ingestion values as a surrogate for sediment ingestion) have been modeled for a 1 to 6-year old as well as for an adult.

Where appropriate, specific exposure assumptions have been adopted from USEPA guidance (1989a, 1990b, 1991a). Where USEPA guidance was either incomplete, not specific to the age groups modeled, or not relevant to this assessment, additional sources of exposure information were used. For the scenarios involving exposure to the MFLBC, site-specific information on current frequency of exposure for this population was obtained from a survey of the MFLBC conducted by the Ohio Department of Health (ODH). In September 1989, the ODH conducted a survey of area residents to determine the extent of exposure to mirex among persons living in the vicinity of the Site and along the MFLBC from Salem to Lisbon. Subjects, chosen based on their proximity to the creek, were obtained by mailing questionnaires to 575 area families who lived on roads that ran adjacent to the creek between Salem and Lisbon, by placing 100 questionnaires in area libraries, and through announcements in three local newspapers and at public meetings. Data on potential exposures were obtained through questions concerning fishing and recreational contact with the creek, consumption of

game and farm products, and employment history. The survey and results of the survey provided to ENVIRON by the ODH are included in Appendix J. Because this survey provides site-specific information on the extent of potential exposure of local residents to the MFLBC, these data were used, as appropriate, in preference to default assumptions recommended in USEPA guidance.

In using these data, consideration was given to certain characteristics and limitations of the survey for use in the context of a risk assessment. Because the study was conducted specifically to identify those members of the local population believed most likely to have been exposed to mirex, the response from study participants is unlikely to be representative of all area residents, but rather most representative of those residents living in close proximity to the MFLBC and with the greatest likelihood of creek contact. It was noted that of 675 distributed questionnaires only 200 families responded, resulting in a response rate of 30 percent. Whether a 30 percent response rate might have introduced any bias in the study outcome cannot be assessed. One limitation of the study identified by ODH was that the exposure information provided by survey respondents, who were most likely adult heads-of-household, may not have represented in all cases the potential exposures of all family members. Despite any limitations in the ODH survey, for purposes of characterizing exposure variables in this assessment, the survey data are considered to provide a reasonable measure of current exposure frequency and have been taken into account in the assessment of exposure to fish, game, and MFLBC sediments in the section of the creek subject to the advisory.

Also considered in the development of exposure assumptions for the recreational population is an ODH advisory in effect against eating fish or wading and swimming in the MFLBC from Salem to Lisbon. In assessing potential current exposures in the area covered by the advisory, the assumption is made that the frequency of activities along the MFLBC has been substantially reduced. In the future, however, and in downstream sections (both currently and in the future), activities along the creek are assumed not to be influenced by the advisory, and exposure frequencies are estimated to be higher.

VIII. RISK CHARACTERIZATION

A. Introduction

Risk characterization is the final step of the public health risk assessment process, as described in Chapter I. In this step, the toxicity values (i.e., SFs and RfDs) for the chemicals carried through the quantitative risk assessment are used in conjunction with the estimated chemical intakes for the modeled populations to estimate both potential carcinogenic and noncarcinogenic health risks. The methodology for deriving quantitative risk estimates is presented in Section B below. Section C of this chapter presents the baseline risk estimates for the hypothetical current use and future use scenarios addressed in this risk assessment.

As discussed in Chapter VII, each scenario was modeled for the RME condition. Based on USEPA guidance (USEPA 1989a, p. 6-5), the RME is used to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures.

It is important for the reader to understand that the risk values estimated in this assessment are not actuarial risks, i.e., they are not risks that have been documented as a result of human exposure to the chemicals evaluated. As discussed in Section I.B. of this baseline risk assessment, The Risk Assessment Process, risk estimates are based on a series of conservative assumptions and, as such, represent an upper bound on risk. The risk values presented below are useful because they can be compared with other risks that have been estimated using the same procedures. Perhaps the most useful application of the quantitative risk estimates that follow is as a means for identifying the most significant potential exposure pathways in terms of potential health risks.

The numerical risk estimates that are presented in this chapter must be interpreted in the context of the uncertainties and assumptions associated with each step of the risk assessment process. The major uncertainties and assumptions associated with this risk assessment are discussed in Section D of the chapter.

Finally, Section E of this chapter summarizes the available human data relevant to the MFLBC and discusses these data in the context of the risk estimates developed in this EA.

B. Methodology for Quantitative Risk Estimation

1. Estimation of Cancer Risks

The numerical estimate of the excess lifetime cancer risk resulting from the modeled exposure to a specific potentially carcinogenic chemical can be calculated by multiplying the chronic daily intake (CDI) by the risk per unit dose, or SF, as follows:

$$\text{Risk} = \text{CDI} \times \text{SF}$$

where:

Risk	=	lifetime probability of developing cancer due to exposure to the chemical evaluated
CDI	=	chronic daily intake, mg/kg/day
SF	=	carcinogenic slope factor, $(\text{mg/kg/day})^{-1}$

The above equation is based on the assumption that the dose-response relationship for relatively low intakes (compared to doses frequently administered to laboratory animals, from which dose-response values are generally derived) is linear (USEPA 1989a), and that risk, therefore, is linearly proportional to dose. According to USEPA guidance (1989a), this assumption of linearity is generally valid only at low risk levels (i.e., when intake is generally low). As risk levels approach or exceed 1×10^{-2}, the linear proportionality between risk and dose tends to deviate. While alternate modeling equations are available to extrapolate carcinogenicity data at higher dose levels, the uncertainty associated with the derived risk parameters probably does not warrant a more refined estimation of risk.

Regulatory agencies generally make the conservative assumption that any internal dose of any chemical classified as being potentially carcinogenic, no matter how small, presents some potential carcinogenic risk to humans. This assumption is based on the hypothesis that a small number of molecular events can produce changes in a single cell that can lead to uncontrolled cellular proliferation and eventually to the development of tumor formation (USEPA 1989a). As discussed below in the section on uncertainties, however, the hypothesis that no threshold dose exists for carcinogens is by no means proven, and may not hold for some carcinogens that do not appear to act directly on genetic material (DNA). In cases of multiple chemical exposures, regulatory agencies

also assume cancer risks to be additive (USEPA 1986b, 1989a). Accordingly, the risk estimates summarized in this chapter are the sums of the risk estimates for all chemicals evaluated in this assessment.

In interpreting the significance of the cancer risk estimates, the reader should consider USEPA policy. The Agency has made it clear that it does not consider any specific cancer risk level as representing an insignificant risk. Instead, the USEPA has adopted a risk range. In the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (40 CFR Part 300), USEPA states that: "For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 10^{-4} and 10^{-6} using information on the relationship between dose and response." In the evaluation of estimated cancer risks developed in this assessment (see below), potential cancer risks are evaluated in light of the range of risks generally regarded as acceptable by USEPA.

2. Estimation of Risks for Noncancer Effects

Unlike the measure of risk used for carcinogens, the measure used to describe the potential for noncarcinogenic toxicity to occur is not expressed as a probability of experiencing an adverse effect. Instead, the numerical estimate of the potential for adverse noncancer effects resulting from exposure to a chemical is derived in the following manner:

Potential for
adverse effects = CDI/RfD

where:

CDI = Chronic daily intake, mg/kg/day
RfD = Reference Dose, mg/kg/day

If the resulting ratio, also referred to as the hazard quotient, is less than or equal to one, it is assumed that the exposed population would not be affected. If the hazard quotient is greater than one, there may be concern for potential noncancer effects. A hazard quotient that is greater than one should not be interpreted to mean that adverse effects will occur because of the uncertainty (safety) factors used in estimating the RfD, and the conservative assumptions used in estimating the CDI that tend to overestimate

exposure. As a rule, however, the greater the value of the hazard quotient above one, the greater the level of potential concern.

As a first screening, the hazard quotients for individual chemicals can be added for any single pathway to estimate the occurrence and severity of toxic effects resulting from exposure to multiple contaminants. The USEPA (1989a) calls these summed quotients the Hazard Index (HI). The HI approach assumes that multiple sub-threshold (below the RfD) exposures could result in an adverse effect and that a reasonable criterion for evaluating the potential for adverse effects is the sum of the hazard quotients. If the HI is less than one, cumulative exposures to the substances of interest would probably not result in adverse effects. If the HI is greater than one, there is an increased potential for adverse effects under the assumed exposure conditions. An HI greater than one, however, does not necessarily indicate that the multiple exposure would harm individuals. According to USEPA (1986b, 1989a), this methodology is most properly applied to substances that induce the same effect on the same target organs. Consequently, application of the HI methodology to a mixture of substances that are not expected to induce the same effect on the same organs would likely overestimate the potential for adverse health effects.

C. Risk Estimates

Tables 34 through 41 summarize the potential lifetime excess cancer risk and hazard index estimates for the chemicals under the current and future use scenarios considered in the baseline risk assessment. Appendix K contains tables that list estimated cancer risks and hazard quotients for each of the chemicals for each of the modeled pathways.

1. Current Use Scenario

a) On-site Trespasser

This scenario modeled exposure of an on-site trespasser to chemicals present in the unfenced portions of the Site via incidental ingestion of soils, inhalation of air, and ingestion of surface water and sediments. Resulting cancer risk estimates and HI values for the on-site trespasser under the current use scenario are presented in Table 34. The total excess lifetime cancer risk associated with these pathways is 2×10^{-6}. Approximately 50 percent of this risk level is attributable to the ingestion of soil containing mirex. The cumulative HI value for the on-site trespasser is 0.08.

b) Worker at Locations Adjacent to Site

This scenario modeled exposure of a worker at areas adjacent to the Site via the incidental ingestion of soils. Resulting cancer risk estimates and HI values for the worker at locations adjacent to the Site under the current use scenario are presented in Table 35. The total excess lifetime cancer risk associated with this pathway is 6×10^{-8}, which is primarily attributable to PAHs in soil. The cumulative HI value for the worker is 0.0003.

c) Resident at Locations Adjacent to Site

This scenario modeled exposure of a resident at areas adjacent to the Site via the incidental ingestion of soils and the ingestion of home-grown vegetables. Resulting cancer risk estimates and HI values for the resident at locations adjacent to the Site under the current use scenario are presented in Table 36. The total excess lifetime cancer risk associated with these pathways is 2×10^{-6}. Approximately 85 percent of this risk is attributable to the ingestion of soil. PAHs and pesticides account for approximately 70 percent of the risk due to this pathway; mirex accounts for the remaining 30 percent. The cumulative HI value for the resident is 0.04.

d) Recreational Visitor

This scenario modeled exposure of the recreational population via ingestion of surface water and sediments, ingestion of fish, and ingestion of game. Because an advisory against fishing, wading, and swimming is in effect in the stretch of the MFLBC upstream of Lisbon Dam, exposures are assumed to differ within and outside the advisory area. Resulting cancer risk estimates and HI values for the recreational visitor under the current use scenario are presented in Table 37. The total excess lifetime cancer risk for the population engaged in recreational activities is estimated to be 6×10^{-6} for areas upstream of the advisory and 5×10^{-6} for areas downstream of the advisory. Essentially all of the excess risk is attributable to the ingestion of fish containing mirex. The cumulative HI values are 0.1 for areas upstream of the advisory and 0.06 for areas downstream of the advisory.

e) Flood Plain Resident

This scenario modeled exposure of a flood plain resident via ingestion of soils, and the ingestion of home grown vegetables. Resulting cancer risk estimates

and HI values for the flood plain resident under the current use scenario are presented in Table 38. The total excess lifetime cancer risk associated with these pathways is 4×10^{-6}. Essentially all of the excess risk is attributable to the ingestion of soil containing mirex. The cumulative HI value for the flood plain resident is 0.3.

2. Future Use Scenario

a) On-site Trespasser

This scenario modeled exposure of an on-site trespasser exposed to all portions of the Site via incidental ingestion of soils, inhalation of air, and ingestion of surface water and sediments. Resulting cancer risk estimates and HI values for the on-site trespasser under the future use scenario are presented in Table 34. The total excess lifetime cancer risk associated with these pathways is 8×10^{-6}. Approximately 90 percent of this risk level is attributable to the ingestion of soil containing mirex. The cumulative HI value for the on-site trespasser is 0.5.

b) On-site Worker

This scenario modeled exposure of an on-site worker via ingestion of ground water, incidental ingestion of soils, and inhalation of air. Resulting cancer risk estimates and HI values for the on-site worker under the future use scenario are presented in Table 39. The total excess lifetime cancer risk associated with these pathways is greater than 1×10^{-2} (i.e., one in one hundred). As noted in Section B.1 above, use of a linear model for approximating risks is valid at low risk levels. Because the modeling approach used provides only an approximation of risk, a more refined extrapolation of carcinogenicity data for risk levels estimated to be in excess of 1×10^{-2} is probably not warranted. This risk is driven by the ingestion of ground water containing volatile organic chemicals (viz., tetrachloroethene and 1,1,2,2-tetrachloroethane). The cumulative HI value for the on-site worker is 147. This value is also driven by the ingestion of ground water containing volatile organic chemicals (viz., tetrachloroethene and 1,2-dichloroethene (total)).

c) On-site Resident

This scenario modeled exposure of an on-site resident via ingestion of ground water, incidental ingestion of soils, inhalation of air, and ingestion of vegetables. Resulting cancer risk estimates and HI values for the on-site resident under the future use scenario are presented in Table 40. The total lifetime excess cancer risk associated with these pathways is greater than 1×10^{-2}. This risk is driven by the ingestion of ground water containing volatile organic chemicals (viz., tetrachloroethene and 1,1,2,2-tetrachloroethane). The cumulative HI value for the on-site worker is 446. This value is also driven by the ingestion of ground water containing volatile organic chemicals (viz., tetrachloroethene and 1,2-dichloroethene (total)).

d) Worker at Locations Adjacent to Site

This scenario, which modeled exposure of a worker at areas adjacent to the Site via the incidental ingestion of soils, is the same as that presented above for the current use scenario (see Table 35). The total excess lifetime cancer risk associated with this pathway is 6×10^{-8}. The cumulative HI value for the worker is 0.0003.

e) Resident at Locations Adjacent to Site

This scenario, which modeled exposure of a resident at areas adjacent to the Site via the incidental ingestion of soils and the ingestion of home-grown vegetables, is the same as that presented above for the current use scenario (see Table 36). The total lifetime cancer risk associated with these pathways is 2×10^{-6}. The cumulative HI value for the resident is 0.04.

f) Recreational Visitor

This scenario modeled exposure of the recreational population via ingestion of surface water and sediments, ingestion of fish, and ingestion of game. Under the future use scenario, the advisory against fishing, wading, and swimming is assumed to be lifted, but risks are still presented for areas above and below Lisbon Dam, the approximate location that the current advisory ends, because the mirex concentrations above and below the dam are markedly different. Resulting cancer risk estimates and HI values for the recreational visitor under the future use scenario are presented in Table 41. The total lifetime cancer risk for the

population engaged in recreational activities is estimated to be 7×10^{-5} for areas upstream of Lisbon Dam and 5×10^{-6} for areas downstream of the advisory. Essentially all of the excess risk is attributable to the ingestion of fish containing mirex. The cumulative HI values are 2 for areas upstream of the advisory, and 0.06 for areas downstream of the advisory.

g) Flood Plain Resident

This future use scenario modeled exposure of a flood plain resident via ingestion of soils, the ingestion of home grown vegetables, the ingestion of beef, and the ingestion of milk. Resulting cancer risk estimates and HI values for the flood plain resident under the future use scenario are presented in Table 38. The total lifetime cancer risk associated with these pathways is 7×10^{-5}. The cumulative HI value for the flood plain resident is 2. Essentially all of the excess risk is attributable to the ingestion of beef and milk containing mirex.

3. Discussion of Risk Estimates

An evaluation of the risk estimates from exposure to chemicals for each of the modeled populations indicates the following:

- No excess cancer risks are above 10^{-4} nor are any HI values above one for any modeled population in the current use scenario. This indicates that the concentration levels present in the study area are acceptable for exposures assessed under the current use scenario.

- Under the future use scenario, excess cancer risks are above 10^{-4} for the on-site worker and on-site resident population. Under these hypothetical future use scenarios, the risks ($> 1 \times 10^{-2}$) would clearly exceed the range of risk deemed acceptable by USEPA. These risks, shown in Tables 39 and 40, are attributable to the ingestion of ground water from an on-site well assumed to be situated at the point where the ground water concentrations are the highest. This situation clearly is a worst case estimate and in no way implies that this scenario is remotely likely in the future.

- Under the future use scenario, the HI values are above one for the on-site worker and on-site resident population. These values (147 for the worker and 446 for the resident) are also driven by the ingestion of ground water from an on-site well.

- Under the future use scenario, the HI values slightly exceed one for the flood plain resident and the recreational visitor upstream of Lisbon Dam (HI value of 2 for both of these populations). These values are driven by the ingestion of beef and milk for the flood plain resident and by the ingestion of fish for the recreational visitor.

As discussed in Section V.C of this EA, some potential pathways of exposure were not assessed quantitatively. This is because of the substantial uncertainty inherent in modeling these pathways, or because consideration of these pathways in a more rigorous fashion would not likely change the overall assessment of risk for the populations evaluated in this EA. For example:

- Potential risks associated with direct contact of on-site and off-site soils and sediments were based on incidental ingestion of soil and sediment. Potential risks associated with dermal contact with these soils and sediments were not assessed quantitatively. As noted in USEPA's *Dermal Exposure Assessment: Principles and Applications* (USEPA 1992e), dose and risk estimates based on the available models for estimating dermal uptake of chemical compounds in soil are considered highly uncertain. Even less is known about dermal uptake from sediments. USEPA Region III has noted that because of this uncertainty and because dermal exposures are likely to be insignificant relative to exposures associated with incidental ingestion of soil, quantitative evaluation of exposures associated with dermal contact with soil are not required. Because incidental ingestion of soil and sediment were assessed quantitatively, the majority of potential exposures and associated risks for chemicals in soil and sediment were likely captured in the EA.

- Potential risks associated with direct contact of on-site and off-site surface water were based on incidental ingestion of surface water. Potential risks associated with dermal contact with these surface waters were not assessed quantitatively. As discussed in Section D.3 of this chapter, exposures associated with dermal contact with water are highly uncertain. Furthermore, potential exposures via dermal

contact are expected to be insignificant. Potential contact with on-site surface water is likely to be limited to occasional contact by trespassers and, because of the size of ditches, to a limited amount of body surface. Therefore, any potential risks associated with dermal contact with drainage ditch water can reasonably be expected to be less than the potential upper bound cancer risk estimated for incidental ingestion of surface water (2×10^{-7}). The potential risks associated with dermal contact with surface water in the MFLBC are not expected to be significant because only three chemicals were detected in MFLBC surface water with relatively low detection frequencies and low (μg/liter) concentrations. Any potential risks associated with dermal contact with water from the MFLBC are expected to be de minimis, based on the potential upper bound cancer risks estimated for incidental ingestion of MFLBC surface water of 4×10^{-9}.

- Under the future on-site scenario, hypothetical worker and resident populations were assumed to use on-site ground water as a domestic water supply. Potential risks associated with this ground water use were based on ingestion of ground water. Potential risks associated with dermal contact with ground water and inhalation of vapors during showering were not assessed quantitatively. As discussed in the Section D.3 of this chapter, exposures associated with dermal contact with ground water are highly uncertain and are likely to be significantly less than exposure associated with the ingestion of ground water. Also, ENVIRON's experience indicates that risks associated with the inhalation of vapors during showering are unlikely to be greater than those associated with ground water ingestion. Based on the above, and the fact that potential risks associated with exposure to ground water under future use on-site conditions via ingestion alone are in excess of 10^{-2} (one in one hundred), consideration of dermal contact with ground water and inhalation of vapors while showering would not change the nature of potential future risks for ground water.

- Potential exposure to chemicals detected in on-site air was quantitatively assessed for on-site receptors. The air data were based on two sampling events, and are thus unlikely to be representative of long-term exposure. No air data were collected at off-site areas. Due to the additional uncertainty associated with modeling off-site air exposures using the on-site data, potential exposures to chemicals present in off-site areas were not assessed quantitatively.

- Under the future on-site scenario, hypothetical worker and resident populations were assumed to use on-site ground water as a domestic water supply. There are no known current exposures to chemicals present in the ground water in the study area. In the absence of any remedial action, off-site migration of chemicals in ground water could occur, which may lead to future off-site exposures. These off-site exposures would be less than those estimated for future on-site populations. Because of insufficient data to model the migration of ground water, these off-site exposures were not modeled quantitatively.

D. Uncertainties and Limitations in the Risk Assessment Process

Risk assessment provides a systematic means for organizing, analyzing, and presenting information on the nature and magnitude of risks posed by chemical exposures. Nevertheless, uncertainties and limitations are present in all risk assessments because of the quality of available data and the need to make assumptions and develop inferences based on incomplete information about existing conditions and future circumstances. These uncertainties and limitations should be recognized and considered when discussing quantitative risk estimates.

In general, the uncertainties and limitations in the risk assessment fall into the following categories:

- environmental sampling and laboratory measurement;
- mathematical fate and transport modeling;
- receptor exposure assessment; and
- toxicological assessment.

1. Uncertainties in Environmental Sampling and Laboratory Measurement

The quality of the analytical data used in a risk assessment depends on the adequacy of the set of rules or procedures that specify how a sample is selected and handled, i.e., the sampling plan (USEPA 1988a). Uncertainties that may be associated with the data include sampling errors, laboratory analysis errors, and data analysis errors. The quality assurance and quality control review procedures used to minimize these uncertainties are described in other parts of the RI.

2. Uncertainties in Mathematical Fate and Transport Modeling

The use of mathematical models to predict the fate and transport of chemicals is well accepted in the professional scientific community and has been widely endorsed by USEPA since it issued its *Superfund Exposure Assessment Manual* (USEPA 1988b). USEPA does not, however, provide specific guidance concerning the selection of specific models from among a wide variety available for a given purpose. Indeed, the trade-off between simplicity, generality, and accuracy is best made by considering the needs and available data of the site in question.

Uncertainty with respect to the vegetable uptake model employed in this assessment is introduced by comparison of experimentally tested plants with common garden vegetables. Specifically, the de La Cruz and Rajanna (1975) study, upon which vegetable uptake of mirex is estimated, was based on plants with fibrous roots that were a mixture of mono- and dicots (e.g., garden beans, soybeans, sorghum and wheat). Such plants may not be exact surrogates for all vegetables commonly grown in gardens, most notably, plants with tap roots or tubers, or non-leafy aboveground produce. The potential effect of this uncertainty on the ultimate risks from vegetable ingestion is unknown.

3. Exposure Assessment Uncertainties

a) General Considerations

In any risk assessment, a large number of assumptions must be made to assess potential human exposure. In the conduct of the exposure assessment, it was necessary to develop assumptions about general characteristics and potential activity or exposure patterns for current and hypothetical future populations in the study area. In developing the future use scenarios, exposure assumptions were made that involved the absence of actions already taken to mitigate exposures to chemicals in on- and off-site media. Specifically, hypothetical future exposures were estimated assuming the absence of a fence around portions of the Site, the absence of a fishing advisory above Lisbon Dam, and the absence of fences and bridges installed along the MFLBC to prevent access of livestock to the creek and its floodplain.

For each exposure pathway modeled, assumptions were made about the number of times per year an activity could occur, the routes of exposure by which an individual could be exposed, the amount of contaminated media to which an

individual could be exposed by the activity, and the amount of chemical that could be absorbed by each route of exposure. In the absence of site-specific data, the assumptions used in this baseline risk assessment are generally those consistent with USEPA guidance for deriving estimates of the reasonable maximum exposure (RME), defined by USEPA as "the maximum exposure that is reasonably expected to occur at a site" (USEPA 1989a).

Recent USEPA guidance, however, presents standard default values that can lead to the RME risk estimates being at the very high end of the risk distribution (USEPA 1990a,b). Risks based on a combination of several upper bound values (i.e., 90th or 95th percentile values) may well be in excess of the 99th percentile exposure and thereby outside the range of exposures that might **reasonably** be expected to occur at a site. To the extent possible, an attempt was made to avoid combining too many upper bound exposure variables in assessing any pathway. Because information on the statistical distribution of exposure variable values is limited, however, it is not always possible to characterize exposure estimates quantitatively.

In two recent guidance documents (USEPA 1992c, 1992d), the Agency has recognized that the RME approach is incomplete by presenting only a point estimate of risk with no understanding of where it falls on the risk distribution. In these documents (USEPA 1992c, 1992d), the Agency suggests that one approach to quantitative uncertainty analysis is the development of a risk distribution. One method for accomplishing this type of analysis is a probabilistic numerical method such as Monte Carlo simulation. A Monte Carlo simulation was not conducted as part of this risk assessment.

b) Qualitative Evaluation of Potential Dermal Exposure

Potential exposures resulting from dermal contact with contaminated soil, sediment, and water were evaluated qualitatively in this assessment relative to the potential exposures estimated quantitatively for incidental ingestion of soil, sediment, and water. As noted in USEPA's *Dermal Exposure Assessment: Principles and Applications* (USEPA 1992e), dose and risk estimates based on the available models for estimating dermal uptake of chemical compounds in soil are considered highly uncertain. Experimental data on dermal absorption from soil relevant to quantitative risk assessment are available for only a limited number of compounds. Even less is known about dermal uptake from sediments. USEPA

Region III has noted that estimation of exposure via dermal contact with soil is highly uncertain and that dermal exposures are likely to be insignificant relative to exposures associated with incidental ingestion of soil. USEPA Region III does not require, therefore, quantitative evaluation of exposures associated with dermal contact with soil.

Given the substantial uncertainty in the estimation of exposures associated with dermal contact with soil and sediment, this pathway was not quantitatively evaluated in the assessment of the Salem site. Because incidental ingestion of soil and sediment were assessment quantitatively, it is expected that the majority of estimated exposures to chemicals in soil and sediment were captured.

Potential exposures to chemicals in surface water and ground water were also evaluated qualitatively by comparison to potential exposures via ingestion of surface or ground water. Like soil, estimates of exposure via dermal contact with water are highly uncertain, and from prior experience, are less significant than exposures associated with ingestion of water. Given the nature of contaminants in surface water and ground water at the Site and risks associated with ingestion of water, it is unlikely that quantitative consideration of dermal contact with water would significantly alter overall risks. Excess upper-bound cancer risks associated with future exposure to contaminants in on-site ground water via ingestion are in excess of 1 in 100 (10^{-2}); any consideration of the potential contribution from dermal contact with ground water would not appreciably change the nature of potential future risks from this medium. Potential contact with on-site surface water is likely to be limited to occasional contact by trespassers and, because of the size of ditches, to a limited amount of body surface. Any exposure via dermal contact with ditch water can reasonably be expected to be smaller than incidental ingestion. Dermal contact with surface water in the MFLBC is not expected to be significant because only three chemicals were detected in MFLBC surface water with relatively low detection frequencies and low (μg/liter) concentrations. In summary, based on the nature of the chemical contaminants in surface water and prior experience that suggests that dermal contact risks will be smaller than those associated with ingestion, it is unlikely that a qualitative evaluation of dermal contact with surface and ground water will result in a substantial underestimation of potential risks via these media.

c) Qualitative Evaluation of Exposures for Off-site Populations Adjacent to the Site

Potential risks to current and future off-site populations adjacent to the Site via inhalation of chemicals in air from the Site and via ingestion of ground water were estimated qualitatively based on potential risks to populations exposed to these media on-site. Because of attenuation of media concentrations with greater distance from the source, the potential exposures are likely to be far smaller than those estimated for on-site populations.

4. Toxicological Assessment Uncertainties

In the great majority of risk assessments, as in the current risk assessment, available scientific information is insufficient to provide a thorough understanding of all the toxic properties of chemicals to which humans are potentially exposed. It is generally necessary, therefore, to infer these properties by extrapolating them from data obtained under other conditions of exposure, generally in laboratory animals.

Experimental animal data have been relied upon for many years by regulatory agencies and other expert groups for assessing the hazards and safety of human exposure to chemicals. This reliance has been supported in general by empirical observations. There may be differences in chemical absorption, metabolism, excretion, and toxic response, however, between humans and the species for which experimental toxicity data are generally available. Uncertainties in using animal data to predict potential effects in humans are introduced when routes of exposure in animal studies differ from human exposure routes; when the exposures in animal studies are short-term or subchronic; and when effects seen at relatively high exposure levels in animal studies are used to predict effects at the much lower exposure levels found in the environment. The methods for dealing with these uncertainties in the toxicological assessments for noncarcinogens and carcinogens is discussed below.

a) Uncertainties in the Characterization of the Toxicity of Noncarcinogens

In order to adjust for uncertainties such as those discussed above, regulatory agencies often base the acceptable daily intake (or for USEPA, the RfD) for noncarcinogenic effects on the most sensitive animal species (i.e., the species that experiences adverse effects at the lowest dose). This dose is then adjusted via the use of safety factors or uncertainty factors to compensate for lack of knowledge regarding interspecies extrapolation and to guard against the possibility that humans are more sensitive than the most sensitive experimental animal species

tested. As indicated by USEPA, the resulting RfD or RfC is a dose likely to be without appreciable risk with uncertainties spanning perhaps an order of magnitude.

b) Uncertainties in the Characterization of the Toxicity of Carcinogens

For many substances that are carcinogenic in animals, there is uncertainty as to whether they are also carcinogenic in humans. While many substances are carcinogenic in one or more animal species, only a small number of substances are known to be human carcinogens. The fact that some chemicals are carcinogenic in some animals but not in others raises the possibility that not all animal carcinogens are human carcinogens, as well as the possibility that not all human carcinogens are animal carcinogens. The finding that relatively few substances are known human carcinogens may be due in part to the difficulty in performing adequately designed epidemiologic investigations in exposed human populations. Regulatory agencies generally assume that humans are as sensitive to carcinogens as the most sensitive animal species. This is a policy decision designed to prevent underestimating carcinogenic risk. In addition, there are several mathematical models available to derive low-dose SFs from high exposure levels used in experiments. The model used by USEPA (and therefore in this risk assessment) is the linearized multistage model, which provides a conservative estimate of risk at low doses (i.e., the model is likely to overestimate the actual SF). Several of the alternative models often predict lower risk at low doses, sometimes by orders of magnitude. Thus, the use of the linearized multistage model ensures a conservative estimate of the SF. The lack of knowledge regarding the validity and accuracy of this model, however, contributes to the uncertainties in cancer risk estimates.

For suspected carcinogens, the normal procedure used by regulatory agencies, and therefore used here for chemicals of potential concern, is to use the 95 percent upper confidence limit estimated by the linearized multistage model. Use of the 95 percent upper confidence limit value rather than the SF that represents the maximum likelihood estimate provides an estimate of the upper bound on risk.

Application of these mathematical low-dose extrapolation models for carcinogens is predicated on the conservative assumption generally made by regulatory agencies that no threshold exists for carcinogens, i.e., that there is some

risk of cancer at all exposure levels above zero.[7] As previously noted, this no-threshold hypothesis for carcinogens is by no means proven, and may not hold for some carcinogens that do not appear to act directly on genetic material (DNA). Mirex has been tested for potential to damage genetic material in a variety of mutagenicity assays and has been shown consistently to be negative for genotoxic potential. Although the mechanism by which mirex causes an increased incidence of tumors in experimental animals is not known with certainty, the available genotoxicity data suggest that mirex does not directly interact with DNA. The apparent absence of genotoxicity of mirex raises the possibility that mirex may have a threshold for carcinogenicity. If this is the case for mirex, the risk assessment methods applied to mirex may substantially over-estimate low-dose cancer risk.

c) Uncertainties Introduced by Estimation of Toxicity Values by Inter-route Extrapolation

For some chemicals evaluated in the risk assessment, route-specific toxicity values have not been derived. Extrapolation of a toxicity value from one route to the other was performed where the effect was based on a systemic effect and not limited to the site of test material administration. Estimation of toxicity values by route-to-route extrapolation introduced additional uncertainty in the toxicity assessment. Values so derived should, at best, be considered approximations of measures of toxic potential.

d) Uncertainties Introduced by Lack of Toxicity Information

In most risk assessments, chemicals are present that cannot be included in the quantitative risk assessment because little or no information on the toxicity of the chemical is available. In the current assessment, 19 of the 144 chemicals (or groups of chemicals) considered in the risk assessment had no toxicity values. As indicated in Chapter IV, none of these chemicals (or groups of chemicals) are considered by USEPA to be carcinogens or are appropriately treated as carcinogens (note discussion of CDDs and CDFs in Section IV.C.1). It is unlikely that failure to consider these substances in the quantitative risk assessment would result in an underestimation of total risk for the exposed populations modeled.

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[7] While this suggests that any exposure to a carcinogen poses some risk of cancer, the probability may be extraordinarily small, so that, for all practical purposes, no risk exists.

E. Comparison of Risk Characterization Results to Human Studies

The Ohio Department of Health (ODH) initiated an investigation of possible mirex exposure in people living in the vicinity of the Site and the MFLBC. The ODH survey and its findings are summarized in Section E.1 below. Section E.2 discusses the findings of this survey in the context of the results of risk assessment for the MFLBC portion of the study area.

1. ODH MFLBC Survey

In September 1989, the ODH conducted a survey of residents living in the vicinity of the Nease Chemical Company Superfund site to determine whether any residents had detectable amounts of mirex in their blood and to more closely examine the environmental pathways that may have contributed to this body burden. The results of this survey are presented in the report entitled "Assessment of Exposure to Mirex Associated with the Nease Chemical Company Superfund Site in Salem, Columbiana County, Ohio" (ODH 1990b).

Subjects for this study were obtained by mailing questionnaires to 575 area families chosen based on their proximity to the creek, by placing 100 questionnaires in area libraries, through announcements in three local newspapers, and at public meetings. Potential for exposure to mirex through various environmental media was assessed by asking about direct recreational contact with the MFLBC, about consumption of locally caught fish or game, about consumption of locally grown farm or garden products, and about employment history. Activities were ranked based on the known likelihood that a pathway would contribute to human uptake. Activities that involved the consumption of potentially contaminated food were ranked the highest; activities that occurred nearer to the Site were ranked higher than similar activities occurring further from the Site. Based on the responses to these activities, respondents were categorized into one of four exposure groups: "high," "medium," "low," or "no potential exposure". Due to budgetary constraints, only some of the households from each exposure category were asked to participate in the collection of blood samples, although all of the households from the "high" exposure group were invited to participate. In addition to the collection of blood samples to analyze for serum mirex levels, the ODH requested that residents participate in a Caffeine Breath Test. The Caffeine Breath Test is a test designed to measure the induction of microsomal enzymes and was undertaken in this study because mirex is a known inducer of microsomal enzymes.

A total of 200 families responded out of the 675 distributed questionnaires (approximately 30%). Although the original intent of the survey was to collect potential exposure information for each individual in the families surveyed, an error in the survey form limited collection of exposure information only to the respondent who completed the form for the entire family. Age, occupational exposure and coincidental consumption of farm animal products were available for all respondents. It appears that each family was ranked based on the response of the family member completing the questionnaire.

Of the responding families, 78 reported no exposure pathways for any family member, and 122 families reported varying degrees of exposure. The most commonly reported activities (from most to least) were: physical contact with the creek (swimming, wading, etc., 40.5% of the responding families); consumption of locally-caught game (32.5%); consumption of fish (17%); irrigation of crops with water from the creek (8.5%); and consumption of animals that had access to the creek (4.5%). Of the 122 households, 48 were selected from the four exposure categories, and 27 households agreed to participate. This represented 42 area residents. Four former Nease employees were among the study participants. In addition, two staff from the ODH gave blood samples. No mirex was detected in these two samples, so their results were not included in the analysis of risk factors. Only some of the 42 participants agreed to participate in the Caffeine Breath Test. The results of the Caffeine Breath Test were never published.

Participants ranged in age from 8 to 75 years old, with both a mean age of 40.4 years. Of the 42 residents, 28 had no detectable levels of mirex in their serum (67%). Of the 14 individuals with detectable levels, 13 (93%) were male. Among those with detectable levels, serum mirex levels ranged from 0.25 ppb to 2.2 ppb, with a mean level of 0.84 ppb and a median of 0.68 ppb. The four persons with occupational exposure to mirex had the highest mean serum level of mirex (mean = 1.34 ppb, range 0.46 - 2.2 ppb). Only one of these former employees had worked at Nease for greater than one year. All but one of the other 10 persons who had detectable levels of mirex were categorized in the high exposure category. Three variables showed a strong association with detectable amounts of mirex in the serum: male gender, former Nease employment, and consuming farm animal products that had contact with the portions of the MFLBC flood plain containing mirex. Although statistical testing could not be conducted due to the lack of individualized exposure data, 16 of the 18 persons who did not have detectable levels of mirex reported exposure to

mirex through one or more of the other activities/pathways that were examined: local fish consumption, contact with creek waters or sediments, consumption of garden products grown on the flood plain, and consumption of locally-caught game.

It is not possible to determine the doses to which individuals in this study were exposed, nor was information collected about when potential exposures to mirex occurred. It is also not possible to extrapolate the findings of this study to other populations. However, the ODH noted that since only persons who had the highest risk of uptake of mirex were selected to participate in this study, "it is reasonable to assume that people with less risk of significant exposure would have lower serum concentrations or no detectable mirex."

2. Comparison of ODH Survey and Risk Assessment Results

As noted in the ODH report, the study does not provide any evidence of widespread human uptake of mirex in the population living in the vicinity of the Site or the MFLBC. The only two activities correlated with detectable blood mirex levels were (1) consuming animal products from animals that probably had access to the MFLBC or its flood plain and (2) work at the Nease Chemical plant. No correlation was found in this study between fishing, contact with contaminated creek sediment, and eating garden products grown in possibly contaminated soil.

In the risk assessment for the MFLBC, the highest estimated risks were found to be associated with the consumption of animal products (beef and milk) from animals assumed to have access to the MFLBC or its flood plain and from consumption of fish from the MFLBC (in the absence of a fishing advisory). The finding of a relatively higher risk associated with consumption of animal products is consistent with the findings of the ODH survey. The absence of detectable levels of mirex in people who consume fish from the creek indicates that this pathway may not be a significant exposure pathway. The absence of mirex blood levels in surveyed individuals who reported eating fish may reflect the effectiveness of the fishing advisory issued in 1987. ENVIRON notes that blood samples were analyzed for mirex in 1989. Because mirex is a stable chemical with a long half-life in the body, one might have expected some detectable levels in individuals who consumed fish from the creek. The stability of mirex is demonstrated by the presence of detectable levels of mirex in the blood of former Nease workers, since their occupational exposure would have occurred prior to 1973.

The absence of a correlation between detectable mirex blood levels and contact with creek sediments and consumption of garden products is consistent with the findings of the risk assessment that these pathways pose relatively low exposures and associated risks as compared to potential future risks associated with consumption of beef and milk.

IX. OFF-SITE ECOLOGICAL RISK ASSESSMENT

A. Introduction

The objective of this off-site ecological risk assessment is to characterize the potential risks to ecological resources from chemical substances that might have migrated from the Ruetgers-Nease Chemical site in Salem, Ohio, (Site) to the Middle Fork of Little Beaver Creek (MFLBC). Potential ecological risk associated with on-site exposures are discussed in Section X. Previously collected data (1985-1987) on sediments, fish, and benthic macroinvertebrates in MFLBC along with results from the 1990 Remedial Investigation (RI) activities were considered in the off-site assessment. This assessment considers measured and modeled estimates of exposure, the available guidance and published information on the environmental fate and toxicities of the chemicals selected for evaluation, and the expected/known habitats and likely species in the area.

Based on site characterization analytical data, past operations at the Site, and the availability of ecotoxicological effects thresholds for chemical substances, the scope of this off-site assessment is focused on mirex and photomirex (see discussion in Section D). The analytical data on chemical substance levels in surface water, sediments, flood plain soils, and fish, and the station descriptions plus field observations on habitats and fish and wildlife species are detailed in the RI Report (ERM-Midwest, Inc. 1993).

This assessment is consistent with the guidance contained in the USEPA environmental risk assessment manual entitled "Risk Assessment Guidance for Superfund, Volume II - Environmental Evaluation Manual" (USEPA 1989b). Additional guidance with regard to ecotoxicological thresholds/criteria for aquatic and terrestrial wildlife species is taken from USEPA, U.S. Fish & Wildlife Service, and other published scientific reports.

The environmental risk assessment is divided into ten sections as follows:

Section A - Introduction
Section B - MFLBC Sampling Program Summary
Section C - Ecological Resource Characterization
Section D - Selection of Chemicals for Evaluation
Section E - Toxicity Thresholds for Mirex
Section F - Selection of Receptor Species for Evaluation

Section G - Exposure Characterization
Section H - Risk Characterization
Section I - Uncertainties in the Analysis
Section J - Recommendations

B. MFLBC Sampling Program Summary

The most recent MFLBC sampling program was conducted by ERM-Midwest, Inc. and is summarized in the RI (ERM-Midwest, Inc. 1993). A total of 22 surface water, 56 fish tissue, 54 sediment, and 28 flood plain soil samples were collected from Feeder Pond, Slanker Pond (located near but northeast of the Site), and a number of locations along approximately 50 miles of the MFLBC from upstream of the Site to near East Liverpool, Ohio. A total of 52 stations were sampled between April 16 and May 21, 1990 (see Figure 2). Station #1 is in the MFLBC, upstream of the Salem municipal wastewater treatment plant (WWTP) outfall. Stations #2 to #5 are downstream of the WWTP, but upstream of where Feeder Creek (which receives drainage from the Site) flows into the MFLBC. Station #6 includes Feeder and Slanker Ponds, which are between the Site and the MFLBC. Stations 19A and 19B were taken from wetland areas adjacent to the MFLBC. Stations #7 through #39 are in the MFLBC upstream of the Lisbon Dam with the exception of Station #29, which is in the Stone Mill Run tributary to MFLBC. Stations #10, #12, #17, #19A, #19B, #27, and #43 also included flood plain samples associated with farms or wetland areas. Stations #40 through #52 are in the MFLBC downstream of the Lisbon Dam, with the exception of Station #47, which is in the West Fork of Little Beaver Creek, and Station #50, which is in the North Fork of Little Beaver Creek.

In addition to the RI sampling, several other MFLBC sampling programs provide data potentially applicable to the evaluation of ecological risks. The Ohio Environmental Protection Agency (OEPA) conducted an aquatic biological survey in 1985. The U.S. EPA conducted a survey of MFLBC benthos and sediments in 1987. The Ohio Department of Health (ODH) conducted a trapping survey of mirex in raccoons and opossums in 1989. The details of these programs are summarized in the RI; relevant results are presented and discussed below.

C. Ecological Resource Characterization

1. Habitat

a) Geographical Overview of the Area Surrounding MFLBC

The MFLBC flows through Columbiana and Mahoning Counties in Ohio. Figure 3 depicts the watershed of MFLBC, as well as major roads and physical features within one mile of the channel.

Mahoning County and the northern half of Columbiana County lie in the geologic area known as the Glaciated Appalachian (or Allegheny) Plateau (Lafferty 1979). The plateau's rolling hill topography is interspersed with cities and villages, and dairy and grain farms. Natural systems, including forests, bogs, old fields, and surface waters have survived because of the physiography of the plateau. Valley areas and the scattered level topographies have been heavily utilized by man, leaving the remaining areas predominantly natural.

The southern half of Columbiana County lies in the Unglaciated Allegheny (or Appalachian) Plateau (Lafferty 1979). Most of the original oak-hickory forests of this area were cut and burned during the eighteenth and nineteenth centuries, during which forestland gave way to agriculture, charcoal production, and coal mining.

The U.S. Forest Service (Dennis and Birch 1981) estimated that approximately 72,000 acres of Mahoning, and 133,100 acres of Columbiana County are forest lands. These areas represent 27 and 39 percent of the land areas of Mahoning and Columbiana Counties, respectively.

b) Wetland Areas

The National Wetlands Inventory (NWI) overlays for the Damascus, Salem, Elkton, and Lisbon, Ohio 7.5 minute quadrangles were used to identify wetland habitats that are closely associated with MFLBC. Figure 4 depicts the locations and types of wetlands listed in the NWI. The figure provides more detail on wetlands upstream of Lisbon Dam than downstream because it is believed that the upstream areas could be associated with greater ecological exposures due to higher concentrations of mirex in the MFLBC above the Lisbon Dam.

c) **Aquatic Habitat**

The MFLBC is a series of shallow riffles and pools with a maximum depth less than 1 meter and with half of the sampling stations less than 0.3 meters in depth. Stream width ranges from approximately 4-8 meters above the Lisbon Dam, to 15-35 meters below. Average stream velocity at the time of sampling (Spring 1990) was less than 0.5 meters per second, with a discharge of 5-40 cubic feet per second (cfs) above the dam, and 100-300 cfs below. The creek substrate varies from bedrock outcrops and rubble-gravel-boulders in some areas, to sand, silt, and clay in the forested and emergent wetlands.

In 1992, OEPA produced a summary of the aquatic habitat characteristics of the MFLBC using data derived from surveys conducted in 1985 (Yoder, 1992, Appendix L). This summary detailed the Qualitative Habitat Evaluation Index (QHEI) for sampling stations along the MFLBC. The QHEI is an index of macro-habitat quality designed to provide a measure of habitat that generally corresponds to those physical factors that affect fish communities and which are generally important to other aquatic life (Rankin 1989).

The QHEI values for each survey station along the MFLBC can be compared to the ranges of QHEI values that have been established for streams within Ohio that: (1) do not attain the classification of a warm water habitat (WWH) (i.e., QHEI < 46); (2) attain the WWH classification (i.e., QHEI 46-60); or (3) attain the exceptional warm water habitat classification (EWH) (i.e., QHEI >60) as presented in Rankin (1989). Table 42 presents the QHEI values that have been calculated for OEPA survey stations on the MFLBC along with the results of comparisons with the WWH and EWH QHEI attainment thresholds. The table indicates that the MFLBC provides WWH or EWH at most stations. The exceptions to this are the OEPA survey stations at river mile 28.8 (corresponding to RI sampling stations #17-18), 26.8 (RI sampling stations #22-23), and 20.9 (RI sampling stations #31-32), which do not meet criteria as warm water habitats. Fewer than half of the areas sampled attain the exceptional warm water habitat designation. Yoder (1991) states that there are possible sources of aquatic community impairment, such as the Salem wastewater treatment plant (WWTP) and a variety of small industries discharging into Buttermilk Creek (i.e., upstream of the Site), that may modify the realization of WWH use attainability within the MFLBC. These modifications would be independent from any contribution from

the Site. The OEPA data are considered and discussed further in Section H.2 below.

d) Wild and Scenic River Status

Figure 5 is a map showing the reaches of the North, Middle, and West Forks and the main stem of Little Beaver Creek that are Federally designated as wild or scenic rivers. All of these areas are below the Lisbon Dam, which is about 30 miles downstream of the Site.

Little Beaver Creek in Columbiana County was designated as Ohio's first wild river in 1974. The 20-mile wild portion of Little Beaver Creek includes: a reach of the West Fork downstream from Y-Camp Road to the confluence with the Middle Fork of Little Beaver Creek; the North Fork of Little Beaver Creek from Jackman Road to the confluence with the main stem of Little Beaver Creek; and the main stem from the confluence of the West and Middle Forks downstream to 0.75 mile north of Grimm's Bridge.

The 16-mile scenic portion of Little Beaver Creek includes: the North Fork from the Ohio-Pennsylvania state line downstream to Jackman Road; the Middle Fork from Elkton Road downstream to the confluence with the West Fork; and the main stem from 0.75 mile north of Grimm's bridge downstream to the Ohio-Pennsylvania state line.

e) Riparian Habitat

Aerial photographs of the MFLBC, taken as part of the RI, were reviewed to identify principal morphological cover types along the riparian zones of the MFLBC. Figure 6 depicts the forested and open field areas immediately adjacent to the creek. Detailed summaries of habitat and wildlife observations made during the RI sampling at each of the 52 stations along the MFLBC are presented in the RI Report. In general, the terrestrial habitat goes from a "scrub/shrub wetland fringe" at station #1 into forested land at stations #2 to #5. Station #6 includes Slanker and Feeder Ponds. Most of the remaining stations above the Lisbon Dam are forested or forest-wetlands with the exception of two emergent-wetland areas which occur at stations #25 and #33 to #34. Below the dam (i.e., stations #40 to #52) much of the riparian habitat is forested; however, no aerial photographs of the MFLBC below the Lisbon Dam were available to confirm this field observation.

2. Species

a) Breeding Birds of the Area Surrounding MFLBC

No surveys were conducted to quantitatively assess the avian communities (or their habitats) along the MFLBC. However, the *Ohio Breeding Bird Atlas* (Peterjohn and Rice 1991) was consulted for a listing of bird species known to breed in Mahoning and Columbiana Counties. Appendix M is a compilation of bird species listed as confirmed, probable, or possible breeders in the two counties.

b) Mammals of Mahoning and Columbiana Counties

No surveys were conducted to quantitatively assess the mammalian communities (or their habitats) along MFLBC. However, Gottschang (1981) was consulted for a listing of mammalian species either confirmed in, or with ranges encompassing, Mahoning and Columbiana Counties. Appendix N is a compilation of these species.

c) Aquatic Organisms

As mentioned previously, OEPA conducted a survey of fish and invertebrate communities of the MFLBC in 1985. The fish and invertebrate species identified at each 1985 sampling station are listed in Appendix L.

More recently, qualitative observations made during the 1990 RI fish tissue sampling confirmed the presence of 12 families and over 50 species of fish in the areas adjacent to the sampling stations. A list of fish species identified during the sampling and their relative abundances at each station are summarized in Section 3.5 of the RI Report. Herbivores, omnivores, and carnivores were represented at nearly all the sampling stations.

The USEPA 1987 survey of benthos in Little Beaver Creek (Metcalf & Eddy 1988) provides more recent data than the 1985 OEPA survey. The most obvious pattern in the data was a higher number of pollution tolerant species (e.g., *Tubificidae* [worms] and *Chironomidae* [midges] based on Plafkin et al. 1989) upstream of the Site but downstream of the Salem WWTP and extending for approximately three river miles below the Salem WWTP. These pollution tolerant species are then replaced by a higher number of pollution sensitive species

downstream of this area (e.g., *Tricoptera* [caddisflies] and *Ephemeroptera* [mayflies], again based on Plafkin et al. 1989).

d) Threatened, Endangered, and Special Concern Species

In 1993, the Ohio Department of Natural Resources, Division of Natural Areas and Preserves conducted a search of the Natural Heritage Data Services inventory of threatened and endangered species for a three-mile wide corridor along the MFLBC (Appendix O). Figure 7 is a map of the MFLBC showing the approximate positions of each plant and animal species location identified in the 1993 search. Three bird species of state special interest, the sharp-shinned hawk *(Accipiter striatus)*, the sora *(Porzana carolina)*, and the virginia rail *(Rallus limicola)*, are probable or confirmed nesters within 1.5 miles of either side of the MFLBC below the Site. The state endangered American bittern (*Botaurus lentiginosus)* is listed as a probable or confirmed nesting bird in the corridor upstream from the Site. One state endangered plant species, the prairie tick-trefoil *(Desmodium illinoense)*, was identified in the corridor in 1960 only. Four state threatened plant species (crinkled hairgrass *(Deschampsia flexuosa)*, pale straw sedge *(Carex albolutescens)*, straw sedge *(Carex straminea)*, and southern woodrush *(Luzula bulbosa)* in 1967 only) were identified in the corridor. Eight state potentially threatened plant species (Long Beech-fern *(Phegopteris connectilis)* in 1960 only, spotted coral-root *(Corallorhiza maculata)* in 1964 only, tall manna-grass *(Clyceria grandis)*, beaked sedge *(Carex utriculata)*, tubercled rein-orchid *(Platanthera flava)* in 1960 only, catberry *(Nemopanthus mucronatus)*, necklace sedge *(Carex projecta)*, and swamp jack-in-the-pulpit *(Arisaema stewardsonii))* were identified in the corridor.

An April 1991 search of the inventory indicated that there have been sightings of a state endangered amphibian, the hellbender *(Cryptobranchus alleganiensis)* near the confluence of the Middle and West Forks of Little Beaver Creek, which is just near the Beaver Creek State Park border downstream of the Lisbon Dam. This species was not identified in the 1993 search of the database.

The Biological Technical Assistance Group (BTAG) has commented that the area surrounding the MFLBC is within the range of the Indiana bat *(Myotis sodalis)*. Distribution maps for the species indicate that Mahoning and Columbiana Counties are within the range of the bat (Gottschang 1981). However, the Ohio Natural Heritage Data Service provided no information on the

current or historical occurrence of this species in the MFLBC area in either the 1991 or 1993 search.

D. Selection of Chemicals for Evaluation

Table 43 lists the chemicals detected in various environmental media either in, or hydrologically adjacent to, the MFLBC. Maximum measured levels of chemicals in water, sediments, and fish, plus available aquatic toxicity thresholds and the rationale for either including or eliminating the chemical in this assessment, are provided in Table 43. The criteria used to select a chemical for evaluation and the results are discussed briefly below.

If a chemical was detected at a frequency of less than or equal to 5 percent in any environmental medium for which there are at least 20 samples, it was not considered further in this assessment (RAGS, USEPA 1989a). This is further justified for ecological assessments based on the premise that significant impacts on individuals will not occur from a rare exposure and that only a very small portion of a population would be exposed at all to infrequently occurring chemicals. Off-site flood plain soil data are not listed in Table 43 because only photomirex and mirex were detected and both of these chemicals are being assessed in all media.

Thirty chemicals were detected in MFLBC sediments. Sediment toxicity screening values were obtained from NYSDEC (1989; proposed criteria for nine of the chemicals in MFLBC sediments) as derived using USEPA (1988c) methodology for calculating sediment criteria. This same USEPA methodology was used to calculate toxicity screening values for 19 of the remaining 21 chemicals detected in MFLBC sediments. The USEPA methodology involves a sediment:pore water partition calculation and use of water quality criteria values for each chemical. For this assessment, either OEPA (first choice) or USEPA water quality criteria values were used. The maximum sediment concentrations for 17 of the 19 chemicals were below their toxicity screening values and were therefore eliminated from further consideration. Two chemicals, mirex and 4-methylphenol had maximums that exceeded their toxicity thresholds. Of the remaining two of 21 chemicals, one (diphenyl sulfone) was detected in less than 5 percent of the RI sediment samples and was therefore eliminated from further consideration. The other chemical, photomirex, was combined with mirex and treated as a single chemical in the exposure assessment.

Only three chemicals were detected in surface water samples, none are considered further in this assessment. One [bis (2-ethylhexyl) phthalate] was detected at levels below USEPA Ambient Water Quality Criteria (AWQC) levels, a second (chloromethane) was

detected at levels below estimated aquatic toxicity thresholds, and the third (diphenyl sulfone) was detected in less than 5 percent of the samples analyzed.

Of the 24 chemicals detected in fish tissues obtained from MFLBC fish, toxicity screening values were available for only five (aldrin, endrin, alpha-chlordane, DDT, and mirex); these screening values were obtained from Newell et al. (1987). The maximum concentrations measured in fish tissue for three of the five chemicals were below the toxicity screening value. The maximum concentration of endrin was about twice the toxicity screening value, but endrin was detected in less than 5 percent of the fish samples. The remaining chemical was mirex.

Based on this analysis, mirex, photomirex, and 4-methylphenol are the only chemicals selected for off-site evaluation because: 1) they were detected frequently in sediment, fish, and soils, 2) there is available ecotoxicology data and guidance for the protection of aquatic species and wildlife, and 3) their maximum detected concentrations exceed available toxicity threshold estimates.

A total of 52 stations were sampled for chemical constituents in surface water, sediments, flood plain soils and fish tissue between April 16 and May 21, 1990 (see Figure 2). A complete list of sample stations, sample types and chemical levels appears in the RI report. Because of their similar environmental behaviors and toxicities, the concentrations of mirex and photomirex have been added together in the data presentation and discussion. They are simply referred to as "mirex" throughout this report.

1. General Results of the 1990 Sampling Effort

The 1990 MFLBC sampling program was conducted by ERM-Midwest, Inc. and is summarized in the RI Report (ERM-Midwest, Inc. 1993). A total of 22 stations (22 total samples) were sampled for surface water, 28 stations (multiple samples taken from each) for fish tissue, 52 stations (54 samples) for sediment, and seven stations (28 samples) for flood plain soils. Appendix P.1 provides information on the mean mirex concentration (mirex + photomirex) in sediments, fish, and flood plain soils per station. Appendix P.2 provides the mean sediment 4-methylphenol concentrations per station.

a) Surface water

Mirex was not detected in any of the surface water samples analyzed (detection limit = 0.000005 mg/l).

b) Sediment

All of the sediment samples were taken directly from MFLBC, except for the samples from stations 6A-6D which are from Feeder and Slanker Ponds (located between the Site and the MFLBC) and from stations 19A and 19B which were collected from wetland areas adjacent to the MFLBC.

Appendix P.1 summarizes the sediment mirex data presented in the RI Report by sample station. Where duplicate samples were available, they were averaged to obtain a mean for the station. Where mirex (or photomirex) was not detected, half the sample quantitation limit multiplied by the quantitation limit multiplier was used. However, sediment samples with reporting multipliers greater than 5 were not included in the calculation of the means. The majority of the multipliers were less than 5. Five samples (i.e., stations 12, 20, 28, and 30) had reporting multipliers of 33.1, 75.5, 274, 21.9, and 26.2, respectively. These multipliers appeared unusually high and were considered outliers that would inappropriately bias the other results (RAGS, USEPA 1989a). These five samples were not included in deriving mean values.

Appendix P.2 summarizes the sediment 4-methylphenol data presented in the RI Report. Sixteen samples were collected upstream of Lisbon Dam and eight downstream. Only six of the twenty-four samples had detectable concentrations (quantitation limits were 0.350-0.550 μg/kg). The mean and maximum concentrations were 0.499 μg/kg and 2.8 μg/kg upstream and 0.453 μg/kg and 2.1 μg/kg downstream, respectively (Tables 28 and 29).

c) Whole Body Fish Tissue

During the RI, an attempt was made to sample both upper and lower trophic level fish species at each of 28 sample stations along MFLBC (and in Feeder and Slanker Ponds). Upper trophic level fish were filleted (skin-on) prior to analysis. Lower trophic level fish were subject to whole body analysis.

For the purposes of this ecological assessment, all fillet data were normalized to represent whole body residues of mirex. For upper trophic level fish, for which only fillet data were available, the fillet mirex concentrations were multiplied by a factor of two to estimate whole body concentrations. The factor of two is based on data presented by Sherman et al. (1992) and accounts for the difference in the lipid content of fillets and whole body samples for upper trophic level fish species such as smallmouth bass and pumpkinseed. For lower trophic fish, whole body

residues were available at most stations. However, there were five stations at which no upper trophic level fish were collected, and therefore fillets were obtained from lower trophic level fish and substituted. The factor of two utilized to adjust for lipid level in upper trophic level fish was not believed to be appropriate for lower trophic level fish because lipid levels are expected to be greater in lower trophic level fish. In order to estimate whole body mirex residues from fillet data in lower trophic level fish, the average lipid levels for the fillet samples (0.7%, n=5) were compared to lipid levels for whole body (3.4%, n=26). The ratio of lipid in whole body vs. skin-on fillets in lower trophic level fish taken from MFLBC is approximately 5 (3.4/0.7).

Therefore, in all cases, fish tissue data represent the mean whole body residue of all fish taken at a given station. Where mirex (or photomirex) was not detected, half the detection limit multiplied by the quantitation limit multiplier was used. Five data points were not included because they represented skin-off fillets from which whole body concentrations could not be estimated (i.e., one from sampling station #7, two from #39, and two from #48).

d) Flood Plain Soil

Seven sampling stations (#10, #12, #17, #19A, #19B, #27 and #43) were sampled for flood plain soils. At each station, two samples were taken from the east bank and two from the west bank. All four samples per station were averaged to obtain a mean concentration per station. As above, concentrations of mirex and photomirex were summed to obtain a value for total mirex. Where duplicate samples were available, they were averaged to obtain a mean. Where mirex (or photomirex) was not detected, half the quantitation limit multiplied by the quantitation limit multiplier was used.

2. Results of 1989 Ohio Department of Health Survey, and Surveys by Ohio EPA (1985) and USEPA (1987)

In the fall of 1989 the ODH sampled fat and blood from raccoons and opossums trapped at nine sites along the MFLBC. The study was designed to determine if animals other than fish had accumulated mirex. Measured levels of mirex in fat ranged from non-detect to 0.053 ppm; the average level was ~0.010 ppm. These data do confirm that there is some exposure to mammals in the area, and mammals are included in the receptor species evaluated in this risk assessment. There are, however, no

available correlations of concentrations in fat or blood to mirex toxicity. These data are therefore not incorporated in the risk assessment.

Samples of fish and sediment were collected by OEPA and USEPA in 1985 and 1987, respectively. In both fish tissues and sediments, mirex concentrations were generally highest near the Site and then decreased with distance downstream. There were a few fish samples from downstream wetland areas (e.g. Egypt Swamp which is east of Salem) that had relatively high (i.e., > 1 ppm) levels of mirex. Although some fish captured downstream of the Lisbon Dam contained mirex (all but one below 0.1 ppm), sediments collected from stations below Egypt Swamp and downstream to the Ohio River did not contain detectable levels of mirex.

3. Assignment of Stations to Reaches to Derive Average Mirex Concentrations

For the purposes of this risk assessment, the 1990 RI data on mirex levels in flood plain soil, sediment and fish tissue along the MFLBC have been separated into three distinct reaches, as shown in Figure 8. This delineation is based on surface water hydrology considerations. The first segment begins near the Site and ends below the large wetland area (Egypt Swamp) located in the vicinity of sampling station #23. This area is anticipated to be one of relatively high sediment deposition and therefore represents a logical delineation point along the MFLBC, based on surface water hydrology. The second delineation point is the Lisbon Dam, another logical point of sediment deposition differentiation along the MFLBC. These reaches also correspond generally with differences in mirex distribution within the MFLBC; mirex concentrations are generally greatest in Reach 1, decrease in Reach 2, and decrease even further in Reach 3.

Table 44 presents average mirex concentrations in sediment, flood plain soil, and fish tissue in each of the three reaches. A per-reach average concentration is used to estimate mirex exposures in mobile species of wildlife because such species can be expected to inhabit an area that encompasses more than one sample station and, consequently, exposures would be to a range of mirex concentrations in a given environmental media. Station-wide mean sediment levels were used, however, to estimate exposures to relatively non-mobile benthic species.

The sediment concentrations of 4-methylphenol do not appear to vary with location on the MFLBC (see Appendix P.2). Average concentrations were similar, approximately 0.5 μg/kg, upstream and downstream of the Lisbon Dam (see Tables 2 and 28).

4. Mirex

a) Properties and Status

Empirical formula:	$C_{10}Cl_{12}$
Molecular weight:	545.5
Water solubility:	1 ppb (freshwater)
Henry's Law Constant:	5.16×10^{-4} atm-cu m/mol
K_{oc}:	$2.4 \times 10^{+7}$ (HSDB 1991)
log K_{ow}:	6.89 (Veith et al. 1979)

Mirex (perchloropentacyclodecane) has been used extensively in pesticidal formulations to control the imported fire ant, and as a flame retardant in electronic components, plastics, and fabrics. In 1978, the USEPA banned the use of mirex as a pesticide, partly because of the hazards it posed to non-target biota. These included delayed mortality in aquatic and terrestrial fauna, adverse effects on reproduction, early growth and development, and high bioaccumulation and biomagnification in the food chain (Eisler 1985).

b) Fate

Mirex is a stable organochlorine compound, being resistant to chemical, photolytic, microbial and thermal degradation. There is evidence, however, for some degradation to monohydro- (photomirex) and dihydro-derivatives, which have biological activities similar to mirex (Eisler 1985). Mirex absorbs very little UV light in the environmentally relevant range of >290 nm. A photodegradation experiment conducted in pure water for six months showed a half-life (i.e., time required for half of the starting material to be lost) of about 1 year (Smith 1978).

Mirex has a low solubility in water, not exceeding 1 ppb in freshwater and 0.2 ppb in seawater. It is highly soluble in fat and accumulates in fatty tissue. Mirex is rapidly adsorbed onto various organic particles in the water column, including algae, and, eventually, is removed to the sediments. With its relatively high K_{oc} value of $2.4 \times 10^{+7}$, mirex adsorbs strongly to organic materials in soil and sediment, and is generally immobile except for movement via erosion (Smith 1978). Mirex is persistent in terrestrial and aquatic soils/sediments. Degradation half-life estimates range to 10 years or more. In biological systems, elimination

half-lives range from 30 days in quail to 130 days in fish, and to more than 10 months in rats (Eisler 1985).

c) Predicted Bioaccumulation

Mirex bioaccumulates in aquatic organisms, with bioconcentration factors (BCF) in the thousands for algae and aquatic invertebrates, and up to tens of thousands or more for fish. A log BCF of 7 was calculated for mirex in Lake Ontario rainbow trout (Oliver and Niimi 1985). Bioaccumulation factors (BAFs) for birds and mammals exposed to mirex in the food chain are generally less than 50 (Eisler 1985). The highest levels of mirex in exposed organisms are present in fat and in eggs which are high in lipids. [NOTE: BCF applies to aquatic organisms and is based on chemical uptake from water only. BAF applies to aquatic or terrestrial organisms and is based on total chemical uptake from all relevant exposure pathways, but particularly via food.]

No data were available for bioaccumulation in invertebrates exposed to mirex in soils or sediments. Therefore, in this assessment, it is necessary to estimate accumulation using a model.

Based on general fugacity concepts, Connell and Markwell (1990) have suggested that bioaccumulation of organic compounds in invertebrates can best be described as a three compartment model involving the sediment/soil, interstitial water, and the biota, in which the partitioning to organisms from water (BAH_w) divided by the partitioning from water to soil/sediment (K_p) approximates the bioaccumulation from soil/sediment to invertebrate. Partitioning of organic compounds between water and sediment/soil is a function of the compound's affinity for soil/sediment organic carbon (usually expressed as the K_{oc} partition coefficient) and the amount of organic carbon in the sediment/soil (expressed as % carbon). Partitioning of organic compounds from water to organisms (actually organism lipid) is a non-linear function of the affinity of a compound for organic material relative to water (usually expressed as the octanol:water partition coefficient or K_{ow}) and the lipid content of the organism (expressed as % lipid).

Connell and Markwell's equation describing the bioaccumulation of a compound from soil/sediment to invertebrate (BAF_s) is as follows:

$$BAF_s = \frac{BAF_w}{K_p} = \frac{(\%\ lipid)(K_{ow})^a}{(\%\ carbon)(K_{oc})}$$

where: ^{a} is the non-linearity constant for bioaccumulation from water.

This fugacity equation yields a soil-to-earthworm bioaccumulation factor (BAF) for mirex of 0.51 using the following values:

(1) K_{ow} 7,762,471 from log K_{ow} of 6.89;

(2) K_{oc} of 24,000,000;

(3) typical earthworm lipid content of 0.85 percent (Rao and Davidson 1980);

(4) assumed Site-area soil organic carbon of 5 percent (Manahan 1991); and

(5) a non-linearity constant (a) for organochlorine compound accumulation from water to earthworms of 1.14 (Lord et al. 1980).

This 0.51 BAF value is similar to the measured average bioaccumulation factor (0.56) for earthworms exposed to soils containing the similarly organophilic compound TCDD (log K_{ow} 6.9) at soil concentrations ranging from 500 to 5,000 μg/kg (Reinecke and Nash 1984). Because bioaccumulation is in part a function of media concentration, it is important to note that the overall average flood plain soil concentration for mirex along MFLBC (i.e., 555 ug/Kg) falls within the experimental range of soil concentrations reported by Reinecke and Nash (1984).

The same fugacity equation yields a sediment/to/benthic macroinvertebrate bioaccumulation factor (BAF) for mirex of 5.6 using the following values:

(1) log K_{ow} of 6.89 (K_{ow} 7,762,471);

(2) K_{oc} of 24,000,000;

(3) average invertebrate insect lipid content of 15 percent (Hanson et al. 1985);

(4) assumed MFLBC sediment organic carbon of 5 percent; and

(5) a non-linearity constant (a) for organochlorine compound accumulation from water to aquatic invertebrates of 1.11 (Markwell et al. 1989).

This 5.6 BAF value for benthic macroinvertebrates is comparable to the measured bioaccumulation factors for chironomid midge larvae exposed to the structurally similar compound kepone (BAF between 3 and 20 for sediments with 12.3 and 1.5% organic carbon, respectively) in a flow-through laboratory system (Adams 1987).

d) Toxicity (non-human)

In short-term (LC_{50}) studies, aquatic organisms are relatively resistant to mirex toxicity. Delayed mortality, however, is frequently seen in aquatic species after extended periods of exposure (Eisler 1985). This delayed toxicity presumably results from the time required for mirex to accumulate to toxic levels. Significant delayed mortality was observed for freshwater and estuarine crustaceans (i.e., crayfish and shrimp) after exposures as low as 0.0001 mg/l in the water (USEPA 1986d). The maximum acceptable toxicant concentration (MATC) determined for sublethal effects is less than 0.0024 mg/l for amphipods based on growth inhibition, less than 0.005 mg/l for bluegills based on growth, 0.034 mg/l for fathead minnows based on impaired reproduction, and greater than 0.034 mg/l for daphnids and midges based on reproduction and emergence, respectively. Other sublethal effects have been observed for algae, invertebrate and fish species (USEPA 1986d).

Birds appear comparatively resistant to mirex. Eisler (1985) concludes in his review that "most investigators agree that comparatively high dietary concentrations of mirex had little effect on growth, survival, reproduction and behavior of nonraptors, including chickens, mallards, quail and red-winged blackbirds." In birds, mortality has generally been reported following repeated exposures at high dietary concentrations (Eisler 1985). For example, 27 percent of mallard ducks died when exposed to 100 mg/kg mirex in the diet for 25 weeks, 50 percent of ring-necked pheasants died when exposed to 1500 mg/kg in the diet for 5 days, and 20 percent of Japanese quail died in 5 days when exposed to 5000 mg/kg mirex in the diet. Some, but not all, investigators have reported reductions in egg hatchability and chick survival following mirex exposure. A summary of repeat-dose toxicity studies of mirex in birds is presented in Table 45.

In studies of mammalian species, mirex has been shown to cause decreased weight gain, liver effects, reproductive impairment, and, at sufficiently high dose levels, mortality. Reported findings from repeat-dose toxicity studies in mammals are summarized in Table 46.

5. 4-Methylphenol

a) Properties and Status (Howard 1989)

Empirical formula:	C_7H_8O
Molecular weight:	108.13
Water solubility:	22.6 g/L
Henry's Law Constant:	9.6×10^{-7} atm-cu m/mol
K_{oc}:	650
log K_{ow}:	1.94

b) Fate

4-Methylphenol is soluble in water, non-volatile and relatively non-sorptive. These properties indicate that it is relatively mobile in some soils and may leach into ground water. It has been shown to biodegrade rapidly in screening studies using soil, sewage, activated sludge, and freshwater inocula (Howard 1989). It degrades rapidly in soil, with one investigator reporting complete degradation in seven days. The photochemical half-life of 4-methylphenol in the atmosphere is

about 10 hours. Biodegradation is expected to be the dominant loss mechanism when 4-methylphenol is released into water. Experimental half-lives are only a few hours in eutrophic water bodies, but degradation may be preceded by a acclimation period ranging from hours to days. Half-lives in an oligotrophic lake, marine waters, and in water/sediment ecocores were 6, <4, and <2 days respectively.

c) Predicted Bioaccumulation

A bioconcentration factor of 18 was estimated using the log K_{ow}. Because of its low log K_{ow} (1.94), 4-methylphenol is not expected to bioaccumulate significantly in organisms.

d) Toxicity (non-human)

In short-term (LC_{50}) studies, aquatic organisms are relatively resistant to 4-methylphenol; 96 hour LC_{50} in fathead minnow = 19 mg/l, 24 hour LC_{50} in carp = 17 mg/L, LD_0 in *Daphnia* = 12 mg/L, and LD_0 in algae = 6 mg/L (Verschueren 1983). Other toxicity data have also been reported; LC_{50}'s of 1.4 mg/L and 22.7 mg/L in *Daphnia* static-renewal and flow-through tests, respectively, approximately 7.7 mg/L in rainbow trout, and 28.6 mg/L and 19 mg/L in fathead minnows in flow-through and static tests, respectively (OEPA 1988).

The 4-methylphenol chronic water quality criteria was established by OEPA (1988). The OEPA utilizes the same method that the USEPA does to derive water quality standards. However, when there are not enough toxicity data to calculate a standard using the USEPA methodology, the OEPA utilizes another, more conservative method referred to as Method #2. For 4-methylphenol, OEPA used Method #2 (OEPA 1991).

The 6.2 μg/L value was derived by using the lowest species mean acute value (SMAV) of 1.4 mg/L (*Daphnia*) to estimate a final acute value (FAV) of 0.280 mg/L (1.4/5). Because no chronic data were available, and therefore an acute-chronic ratio could not be estimated, the FAV (0.280 mg/L) was divided by a factor of 45 to arrive at a value of 0.0062 mg/L or 6.2 μg/L.

E. Toxicity Thresholds

Toxicity threshold estimates were calculated for those environmental media that represent exposure pathways for mirex. An estimate for an aquatic toxicity threshold for sediment-sorbed mirex is developed in Section E.1 following the general guidance for establishing "interim sediment criteria" (USEPA 1988c). Estimates of chronic toxicity thresholds for mammals and birds exposed to mirex via foodchain pathways are developed in Section E.2 based on a review of the available toxicity literature.

Water quality criteria for mirex are available from both USEPA and OEPA; one part per trillion which is based upon the lowest observed effect level for several crustacean species divided by an uncertainty factor of one hundred. However, since all of the MFLBC surface water samples were below detection for mirex, the AWQC is not needed for this ecological risk assessment.

1. Sediment

The USEPA (1988c) has established a procedure for using the chronic AWQC and the K_{oc} value of a compound to derive an interim sediment quality criterion for the protection of aquatic life. These criteria, which are estimated toxicity thresholds, have been derived by USEPA for only a few organic chemicals; mirex is not one of them. This approach is based upon the assumption that the sediment interstitial water concentration of a compound is the bioavailable and potentially toxic fraction for benthic organisms. This fraction is dependant upon the partitioning between water and sediment organic carbon. The formula for the application of the approach is as follows:

$$\text{Sediment Quality Criterion } (\mu g/g \text{ carbon}) = (\text{AWQC } \mu g/L)(K_{oc})/1000 \text{ g/kg}$$

This calculation yields a estimated sediment "quality criterion" for mirex of 24 μg/g carbon for the AWQC of 0.001 μg/L and a K_{oc} value of 24,000,000.

Assuming an average sediment organic carbon concentration of 50 g/kg (5%), the estimated value for mirex in the MFLBC sediment would be 1,200 μg/kg (24 μg/g C X 50 g C/kg). This value is only an estimate, and because it is based on USEPA's acknowledged conservative assumptions, it should be considered as a screening tool rather than as a definitive toxicity threshold for aquatic species exposed to mirex in sediments.

Sediment toxicity threshold estimates were derived for 4-methylphenol using the same method as for mirex. The USEPA equilibrium partitioning model, a K_{oc} value of 650 (Howard 1989), and the Ohio Warm Water Habitat Criterion (6.2 μg/L) for the chemical (OEPA 1991). The resultant sediment toxicity threshold estimate for 4-methylphenol is 202 μg/kg.

2. Wildlife Foodchains

For purposes of this assessment, mirex toxicity thresholds have been developed for wildlife (birds and mammals) that may be exposed via consumption of food, soils, and/or sediments containing mirex. The thresholds have been developed using the conservative assumption that 100 percent of the diet will contain mirex. The threshold estimates should be considered as a screening tool rather than as a definitive toxicity threshold for wildlife exposed to mirex via the foodchain.

The thresholds were developed using an uncertainty factor scheme developed by Newell et al. (1987) of the New York State Department of Environmental Conservation for the estimation of fish flesh criteria for piscivorous wildlife. These uncertainty factors are applied to no-observed-adverse-effect levels (NOAELs) or to lowest-observed-adverse-effect levels (LOAELs) to account for interspecies variation in sensitivity to a given chemical and for limitations in the available experimental data. The uncertainty factor scheme of Newell et al. (1987) is as follows:

Uncertainty Factors (UFs)

Interspecies Adjustment

- If 3 or more species NOAELs in a class exist, UF = 1
  (i.e., the lowest NOAEL can serve as an estimate of a wildlife NOAEL)
- If 1 or 2 species NOAELs in a class exist, UF = 0.1

Short-term Versus Long-term Adjustment

- For chronic studies (i.e., greater than 90 days), UF = 1
- For short-term studies (i.e., 30-90 days), UF = 0.1

LOAEL to NOAEL Adjustment

- For NOAELs, UF = 1
- To estimate a NOAEL from a LOAEL, UF = 0.2

a) **Birds**

A review of available subchronic and chronic toxicity data for birds is presented in Section D.4.d and Table 45. The edible tissue threshold for birds is based on the study by Kendall et al. (1978). Kendall et al. exposed bobwhite quail to mirex in feed at concentrations of 0, 20, or 40 ppm beginning at one day of age through the grow-out and egg-laying periods. At least 5 breeding pairs per dose group (F_0 generation) were carried through breeding. At four bi-weekly intervals, eggs were incubated. Hatchability and chick survival were determined. The F_0 generation was sacrificed at 36 weeks, and one replicate of F_1 hatchlings receiving dietary mirex was grown out for investigation of reproductive potential of second generation treatment birds. Because of predation in the F_1 generation, only the 1 ppm F_1 group was carried through breeding. The investigators reported no treatment-related effects on productivity, on survival of quail embryos to 3 weeks, egg hatchability, chick survival (to 2 weeks), or survival of the F_1 generation through grow-out and egg laying. Only the 1 ppm F_1 generation group was available for the egg production phase; no problems with embryonation, embryo survival, and hatchability were detected, though data were limited.

Given the absence of adverse effects in any of the treatment groups, the highest dose level tested in the Kendall et al. (1978) study (40 ppm in the diet) is considered to be the NOAEL. This NOAEL of 40 ppm is consistent with the findings of studies in other bird species. Hyde (1972) reported effects on duckling survival at 100 ppm mirex in the diet, but not at 1 ppm. In other studies of mirex in birds, effects have been observed only at dietary concentrations above 100 ppm.

An edible tissue threshold for birds was derived from the NOAEL of 40 ppm using the uncertainty factor scheme of Newell et al. (1987). An interspecies adjustment of 1 was used because at least three species NOELs in birds were reported in the literature. Because the Kendall et al. (1978) study was of chronic duration (i.e., > 90 days) and identified a NOAEL, no additional adjustments (uncertainty factors) need to be applied. Therefore, the fish flesh threshold for birds is estimated to be 40 ppm (mg/kg) in the diet.

For purposes of the risk assessment, a threshold value in units of mg mirex per kg body weight per day (mg/kg/day) was also calculated. According to USEPA 1986 (HED, Standard Evaluation Procedure, Ecological Risk Assessment), the body weight and food consumption rate for the bobwhite quail is 0.17 kg and

0.0152 kg/day, respectively. The toxicity threshold, in mg/kg/day, can therefore be calculated as:

$$\frac{40\ mg/kg\ diet\ x\ 0.0152\ kg\ diet/day}{0.17\ kg} = 3.6\ mg/kg/day$$

b) Mammals

A review of available subchronic and chronic toxicity data for mammals is presented in section D.4.d and Table 46. Based on this review, the reproductive toxicity study of Chu et al. (1981) was selected as the basis for the toxicity threshold for mammals.

Chu et al. (1981) fed groups of rats diets containing 0, 5, 10, 20, or 40 ppm mirex for 13 weeks prior to mating, during a two-week mating period, and through gestation and lactation. Toxicity in adult females was assessed based on hematologic analyses, serum chemistry, liver enzymes and histopathology. Pups were evaluated with respect to body weight (at birth, 4, and 21 days), survival, and histopathology at 21 days. Adult females at 40 ppm showed a significant decrease in weight gain. Litter size was significantly decreased in all treatment groups, and 21-day pup survival was affected at 20 ppm. Enlarged livers were observed in 40 ppm adults. Histopathological changes in the livers and thyroids of mothers and pups were observed in all treatment groups, and cataract formation was reported in pups at 5 ppm. The LOAEL in this study is considered to be 5 ppm; a NOAEL was not identified

The Chu et al. (1981) study was selected as the basis for the toxicity threshold because reproductive endpoints are one of the more relevant to an assessment of ecological risk. Other chronic toxicity studies, which generally evaluated histopathologic lesions associated with mirex exposure, suggest that the lowest reported NOAELs for mirex are about 1 to 2 ppm in the diet, consistent with the LOAEL from the Chu et al. study of 5 ppm.

A toxicity threshold for mammalian wildlife in units of mg/kg/day was derived from the LOAEL of 5 ppm reported by Chu et al. (1981) using dose conversions (ppm diet to mg/kg/day) provided in IRIS (1992) and the uncertainty factor scheme based on Newall et al. (1987). According to IRIS (1992), the dietary concentration of 5 ppm in the Chu et al. study is equivalent to a dose of 0.5 mg/kg/day. An interspecies adjustment of 1 was applied to this LOAEL

because at least three species NOAELs have been reported in the literature (including rats, mice, and dogs, see IRIS 1992). Because the lowest test dose was a LOAEL, an adjustment factor of 0.2 is applied. Thus, the resulting toxicity threshold can be calculated as follows:

$$0.5\ mg/kg/day\ x\ 0.2\ =\ 0.1\ mg/kg/day$$

F. Selection of Receptor Species for Evaluation

Receptor species are those species that are chosen to represent the larger biological community in the risk characterization. Selection criteria include: species which can reliably be determined to be part of the community; species that have a particular ecological, economic, or aesthetic aspect in the area; and species that can, because of toxicological sensitivity or potential exposure magnitude, be expected to represent the potentially most sensitive populations within the local area. Receptor species selection for this off-site assessment involved consideration of the following information:

- The available chemical analysis of media suggest that exposures to sediment, flood plain soils, and fish tissue (or other aquatic organisms that are consumed) are likely to be the predominant sources of mirex in the diets of organisms along MFLBC;

- The available physical/chemical data on mirex indicate some potential for bioacumulation from sediments or soils to invertebrate/small mammal species that are components of the food for upper trophic levels;

- The available data on birds and mammals of Mahoning and Columbiana Counties;

- The potential availability of riverine/wetland/riparian habitats suitable for piscivorous birds and mammals, as well as raptors with such habitat requirements;

- The potential flood plain habitats which may be feeding areas for terrestrial mammals and songbirds;

- Records of sightings of any threatened, endangered, or special concern species; and

- The availability of data concerning animal behavior and potential toxicological response to mirex exposure.

The following receptor species have been chosen for exposure modeling and risk characterization at this Site [NOTE: Aquatic indicator species (e.g. fish and macroinvertebrates) were not chosen because the aquatic biota are dealt with on a community level via biotic indicies.]:

- Great blue heron (*Ardea herodias*) - A piscivorous wading bird susceptible to mirex exposure from consumption of fish and incidental sediment ingestion. Although the blue heron is a consumer of fish, invertebrates, amphibians, reptiles, and small mammals (Martin 1951), an assumption in the initial exposure modeling was made to consider fish as the only dietary component. This assumption allows measured biological residue data in fish and sediment to drive the exposure assessment and is conservative in that it maximizes potential heron exposures to aquatic species shown to accumulate mirex from the MFLBC.

- Belted kingfisher (*Ceryle alcyon*) - A diving bird which feeds primarily on fish, although invertebrates, reptiles, and amphibians have been observed to be components of the diet (Martin 1951). The kingfisher is susceptible to mirex exposure from consumption of fish.

- Sora (*Porzana carolina*) - A wetlands and riparian bird that feeds on aquatic invertebrates (Terres 1980). This species is susceptible to mirex exposure from consumption of mirex bioaccumulated in invertebrates from sediments and incidental ingestion of sediments. This species of state special concern is a known inhabitant of at least one area along the MFLBC.

- Virginia rail (*Rallus limicola*) - A wetlands and riparian bird that feeds on aquatic invertebrates (Terres 1980). This species is susceptible to mirex exposure from consumption of mirex bioaccumulated in invertebrates from sediments and incidental ingestion of sediments. This species of state special concern is a known inhabitant of at least one area along the MFLBC.

- American robin (*Turdus migratorius*) - A songbird that feeds upon terrestrial invertebrates such as earthworms (Martin 1951). The robin is susceptible to mirex exposure from consumption of mirex bioaccumulated in invertebrates from floodplain soils and incidental ingestion of floodplain soils. Although a significant proportion (i.e., up to 80% seasonally) of the robin diet is vegetative matter (Martin 1951), this exposure assessment conservatively assumes the entire diet is from earthworms.

- Northern harrier (*Circus cyaneus*) - A raptorial bird feeding in areas of wetlands and uplands. Craighead and Craighead (1969) indicate that this species feeds primarily on small mammals. It is susceptible to mirex exposure from mirex bioaccumulated in herbivorous and insectivorous small mammal prey from floodplain soils.

- Red fox (*Vulpes fulva*) - A terrestrial predator. Although the red fox consumes terrestrial invertebrates and plant material, the majority of the diet is composed of small mammals (Martin 1951). The fox is susceptible to mirex exposure from mirex bioaccumulated in herbivorous and insectivorous small mammal prey from floodplain soils and from incidental ingestion of floodplain soils.

- Mink (*Mustela vison*) - A mammalian predator of both riparian and upland areas that feeds upon fish, small mammals, invertebrates, and other animal matter (Chapman and Feldhamer 1982). This species is susceptible to mirex exposure from consumption of fish, mirex bioaccumulated in invertebrates from sediments, and mirex accumulated in the tissues of herbivorous and insectivorous small mammals from floodplain soils.

Exposure modeling and risk characterization were not performed for the endangered Indiana bat (*Myotis sodalis*) because there is uncertainty regarding the presence of this species along the MFLBC. Information on habitat requirements of this bat indicate that foraging colonies exist in closed canopy riparian forests of small streams, and that nesting colonies occur in riparian area trees with exfoliated bark and in hollow limbs of dead or dying trees in riparian areas (Chapman and Feldhamer 1982). Consideration of: (1) the bat's dietary components (primarily emergent insects from aquatic systems); (2) it's high daily dietary intake (almost 50% of body weight); (3) the potential for larval aquatic insects to

bioaccumulate mirex from sediments; and (4) the low dietary threshold of effects from mirex in mammals suggest that toxicologically significant exposures of this species to mirex could occur. However, because no habit evaluation has been performed to determine the suitability of MFLBC as habitat for this species, the risk that mirex in MFLBC may pose to the bat cannot be determined.

Direct exposure modeling and risk characterization were not conducted for the Ohio special concern species the sharp-shinned hawk (*Accipiter striatus*). However, the results for the northern harrier can be used as a conservative exposure and risk characterization estimate for the hawk. The sharp-shinned hawk is an accipiter (i.e., a bird hawk) which, by analogy to other accipiters, is primarily a consumer of woodland birds in upland areas (Craighead and Craighead 1969). In fact, the area shown on Figure 7 that is the confirmed sharp-shinned hawk sighting is an upland area. In the MFLBC area, the sharp-shinned hawk's preference for upland birds would very likely result in a lower potential mirex exposure than for a species such as the northern harrier which hunts wetland areas.

G. Exposure Characterization for Receptor Species

Appendix Q presents the exposure models for the eight terrestrial/avian receptor species. The mean media concentrations of mirex in sediment, floodplain soils, and fish were incorporated in the exposure models as indicated. Table 47 presents the estimated daily dose of mirex to each receptor species for the three MFLBC reaches delineated by stations 1-23, 24-39, and 40-52. It should be noted that the values in Table 47 are likely to be conservative overestimates of the daily exposure. Conservative assumptions regarding maximization of the proportions of dietary components likely to contain mirex have been made. In addition, the models conservatively assume that populations of the receptor species will only derive food from the main channel and associated wetlands/floodplains of the MFLBC. Inspection of the map of the MFLBC (Figure 3) reveals that there are a significant number of small tributaries and geographically close open water bodies such as ponds and reservoirs that are upgradient from the main channel of the MFLBC, and, consequently, not likely to contain mirex. It is highly probable that mobile species such as mink, great blue heron, and northern harrier could utilize these alternate habitats with sufficient frequency to significantly dilute any dietary exposure to mirex from MFLBC. Similarly, the robin and red fox are likely to use adjacent upland habitats that are above floodplain influence with sufficient frequency to significantly reduce potential dietary exposures to mirex from the MFLBC.

H. Risk Characterization

To characterize ecological risks, ecotoxicity information and exposure data are integrated to estimate the likelihood that adverse effects may occur. The available data for the MFLBC, combined with the information and guidelines available from the USEPA and other published literature, allow the use of a simple, screening-level quotient or ratio approach for the assessment of potential risks to wildlife. Using the quotient approach, estimated exposures are compared to toxicity thresholds. If exposure is predicted to be less than the toxicity thresholds, risk potential is considered minimal. If exposure is predicted to exceed toxicity thresholds, then the risk potential is greater and further refinements of the exposure and toxicity information may be warranted. This approach does not establish the existence of actual ecological impacts. Rather, the quotient method of risk characterization is an attempt to address the questions of what exposure pathways exist, what receptor populations could be at risk, what is the likely magnitude of the exposure, and what are the severity and time frame of the potential ecological effects. It is important to note that the quotient method utilized in this assessment relies heavily on the toxicity data for mirex effects in individual organisms. Because toxicological sensitivities can be expected to vary from individual to individual, and effects on single individuals may not always impact the overall health of a species population, the quotient method, as it is employed here, may well result in an overestimation of potential risk to receptor species populations within the area surrounding the MFLBC.

1. Risks to Avian and Mammalian Wildlife

To characterize the risk to avian and mammalian receptor species, estimated mean daily exposures (Table 47) were compared to the toxicity thresholds (Section E) of 3.6 mg/kg/day (3,600 μg/kg/day) and 0.1 mg/kg/day (100 μg/kg/day), respectively. Table 48 shows these comparisons. The results of these comparisons indicate that, for each reach of the MFLBC, the mean mirex levels measured in sediments, flood plain soils, and fish or other aquatic organisms do not pose risks to individuals of the six avian receptor species. If individual organisms are not predicted to be at risk, it can be assumed that the population of which that individual is a member is also not at risk. Similarly, the results indicate no risk to the red fox and none to the mink in two of the three reaches. Table 48 does show a slight exceedance of estimated exposure versus chronic toxicity threshold to mink in Reach 1, defined as MFLBC sampling stations #1-23. The exposure estimated at this reach exceeds the toxicity threshold by a factor of 1.5. Given the conservativeness built into the exposure and toxicity assessments, this

value should not be considered different from 1. In fact, Newell et al. 1987, in their discussion of risks to wildlife from mirex and other chemicals, suggest that exceedances of toxicity thresholds of less than 10 are not likely to equate to a high risk potential. Thus the results of this comparison for mink suggest a minimal risk potential. In addition, if there are mink inhabiting the Reach 1 area, and if individual mink utilize the upgradient tributaries and other water bodies adjacent to MFLBC where there is no mirex, then the potential for exposure and risk would be decreased.

2. Risks to Aquatic Organisms

Two approaches are available to characterize the potential risk of mirex to aquatic species that may be exposed in the water column and/or sediments of the MFLBC. They are the quotient method of comparing exposures to toxicity thresholds (as was done for the terrestrial/avian species), and the community analysis approach, which is based on biomonitoring in MFLBC looking for the presence and overall condition of the species. This second approach should provide a better indication of the actual condition in the MFLBC; however, it is often difficult to attribute impacts to the presence of a particular chemical if there are other physical and/or chemical limiting factors in the area.

a) Quotient Method

The risk of mirex in sediment to benthic organisms was evaluated by comparing the mean mirex concentrations at each sediment station with the estimated sediment quality criterion, 1,200 μg/kg, which was developed previously at an assumed 5 percent sediment organic carbon level. Table 49 summarizes the results of this comparison. The table indicates that there is no risk from mirex to benthic organisms at 51 of 52 stations. Only station #10 shows an exceedance of exposure over toxicity, and it is by a factor of 1.4. Given the relatively conservative assumptions built into the sediment criterion derived using the USEPA approach, this degree of exceedance is probably not significant with respect to benthic or other aquatic community impacts.

The risk of sediment concentrations of 4-methylphenol to benthic organisms was evaluated by comparing the sediment concentrations at each of the seven sample stations where 4-methylphenol was detected to the derived sediment toxicity threshold estimate (i.e., 202 ug/L). Four of the seven sample stations showed exceedances. They are: stations #3 (downstream of the WWTP and

municipal landfill), #13 and #15 downstream of the Site but upstream of Lisbon Dam, and #44 downstream of Lisbon Dam. The exceedances were 8, 4, 14, and 10, respectively. These exceedances indicate that there is potential 4-methylphenol exposure risk to benthic organisms in at least some areas of the MFLBC. Exposures were below the toxicity threshold estimates at stations #1, #2, and #42.

A quotient-based assessment of the risks to surface water exposure is not relevant because mirex was below the detection limit in all of the MFLBC surface water samples.

b) Community Analysis

The Ohio EPA has conducted two studies (1985 and 1987) in the MFLBC looking at the aquatic habitats and communities of organisms in the MFLBC. Appendix L contains interpretations of these studies (Yoder 1991, 1992) as well as summaries of the biocriteria values for the Index of Biological Integrity (IBI) and the Invertebrate Community Index (ICI) that were developed for the dataset. The IBI is a biological index that considers fish community structure. The ICI is a biological index that considers the community structure of benthic macroinvertebrates.

IBI values, that is the fish community, are below both the Warm Water and the Exceptional Warm Water Habitat Biocriteria for nearly all the MFLBC survey stations. This includes the sections of the MFLBC upstream of the Site yet immediately down stream of the Salem WWTP. From just above the town of Lisbon down to the confluence of the West Fork with the MFLBC (the furthest downstream station), the IBI improves considerably. The ICI values, that is the invertebrate community, are also below both the Warm Water and Exceptional Warm Water Habitat Biocriteria at most of the sampling stations above the town of Lisbon. There is no apparent correlation between the areas of ICI impairment and mirex in sediment. The IBI impairment above the Site and the lack of a correlation between mirex in sediment and ICI impairment suggest that factors other than mirex are contributing to the biological impairment of the MFLBC.

Yoder (1991) states that the most severe impacts on the MFLBC occur immediately below the Salem WWTP. In addition, Yoder (1991) states that the metric scores of IBI and ICI strongly suggest sewage enrichment, although some metrics are also characteristic of toxicity. Indeed, a review of the summary table

of chemical/physical parameter violations for each river mile in the 1985 Ohio EPA study (see copy of Table 37 from the Ohio EPA data in Appendix 1) shows: (1) dissolved oxygen violations for river miles 40.3, 38.3, 37.7, 36.7, 35.4, and 32.0; (2) iron violations at river miles 40.3, 38.3, 37.7, 35.4, 32.0, 28.8, 27.8, 25.1, 21.8, 20.9, 15.1, 11.0, and 9.0; and (3) ammonia violations at river miles 37.7, 36.7, 35.4, 32.0, and 28.8. Such violations more closely match the observed ICI impairments than do mirex concentrations in sediment. In addition, low dissolved oxygen and high ammonia are classic examples of municipal wastewater enrichment indicators.

Because the analyzed sediment samples for 4-methylphenol are so widely spaced along MFLBC, there is insufficient resolution for the development of any meaningful correlations between sediment concentrations of 4-methylphenol and community level impacts on benthic organisms in the MFLBC.

3. Conclusions

This risk assessment is not based on a data set that was intended to establish the existence of actual ecological impacts, but rather to characterize the potential risks based on the available exposure, toxicological, and ecological information. The overall analysis of available information shows:

1) Based on a screening analysis of the RI sampling data for the 52 MFLBC stations, mirex and photomirex are the only chemicals that were carried through the entire ecological risk assessment process. Concentrations of mirex and photomirex are summed and referred to as "mirex" in the assessment.

2) There are no significant risks predicted for mammals or birds that would be exposed to mirex via foodchain pathways originating from mirex in the MFLBC surface water, sediments, flood plain soils, fish, or other sources of food. There is, however, insufficient information on the likely presence of the Indiana bat in the MFLBC area to characterize potential risk.

   - Populations of piscivorous birds, such as the great blue heron and belted kingfisher, are not expected to be at risk from consumption of mirex in fish or sediments from any of the three defined reaches of the MFLBC;

- Populations of mink are not expected to be at risk in the MFLBC. Although the risk characterization shows that mirex exposures in Reach 1 (sampling stations #1-23) could exceed the toxicity threshold for mammals by a factor of 1.5, this is not considered to represent a significant risk potential. This is because: 1) the risk characterization is based on conservative assumptions; 2) there is a relatively large amount of upgradient aquatic habitat adjacent to Reach 1 that would tend to reduce exposures to mirex for a mobile predator like the mink; and 3) the toxicity endpoints, which are based on affects on individuals, may overestimate affects on populations. In addition, exposures exceeding the conservative toxicity threshold by a factor less than 10 are, according to Newell et al. (1978), not expected to be related to high risk potentials.

- Populations of raptors, such as the northern harrier, and predatory mammals, such as the red fox, are not expected to be at risk via consumption of mirex in flood plain soil and/or small mammalian prey (mice and voles) that feed in any of the three defined reaches of flood plain of the MFLBC;

- Populations of sora and Virginia rail (special concern species) are not expected to be at risk from consumption of mirex from the sediments and from consumption of invertebrates residing in the sediments of any of the three defined reaches of the MFLBC;

- Populations of songbirds, such as the American robin, are not expected to be at risk from consumption of mirex in the flood plain soils or from consumption of soil invertebrates from the floodplain soils of any of the three defined reaches of the MFLBC;

3) There are no significant risks predicted for aquatic species (i.e., fish or benthic macroinvertebrates) as a result of exposure to mirex in the water column or in sediments. In addition, there is no apparent correlation between impairments of fish and invertebrate community structures that were observed by OEPA and USEPA in their 1985 and 1987 biosurveys and the pattern of

mirex distribution in MFLBC sediments. However, indicators of municipal wastewater enrichment (dissolved oxygen and ammonia) appear to be closely associated with areas of observed low invertebrate community metric scores.

4) Results of the quotient method analysis suggest that there may be risks to benthic organisms from 4-methylphenol in some portion of the MFLBC sediments. Measured concentrations at four stations (including upstream of the Nease Chemical Site, downstream of the Site, and downstream of Lisbon Dam) exceed estimated sediment toxicity threshold values by factors of 4 to 14 fold. Considering the conservative assumptions that are part of the assessment, in particular the OEPA method for deriving the water quality standard of 6.2 ug/L, the magnitude of the risk from 4-methylphenol in sediments may be overestimated by the exceedance values. Based on the reported rapid biodegradation of 4-methylphenol in water, sediment, and soils, levels of exposure and therefore risk should decrease in the absence of inputs to the MFLBC.

I. Uncertainties in the Analysis

As is generally recognized by both the scientific and regulatory communities, the actual ecological risk associated with exceeding, for example, a calculated toxicity threshold level for wildlife is subject to all of the assumptions that are used in an extrapolation from available literature data to the site specific situation under assessment. It is generally agreed that some level of extrapolation is acceptable for a baseline risk assessment and that risk management decisions can be made without necessarily having all site-specific data.

The goal of this endangerment assessment is to characterize the potential risks to the environment of chemical substances, specifically mirex and photomirex, that may have migrated from the Site to the MFLBC. A combination of site-specific data and literature information was used in the analysis. The quotient method was used to provide the best available level of quantitation to the analysis. Some of the limitations and uncertainties in the analysis are discussed as part of the risk characterization (Section H - above). The more general uncertainties that are inherent in the quotient method approach for estimating risk potential are summarized below:

1. Toxicity Thresholds

The chronic toxicity threshold criteria for water, sediment, or dietary exposure are estimates based on the results of laboratory tests with "representative species" which are not necessarily those species associated with the MFLBC. By design, the criteria are intended to protect the most sensitive species, and they have conservative application and uncertainty factors built into the calculations from the outset.

2. Exposure Estimates

It is assumed that the sensitive species are exposed continuously and over long periods of time to toxic levels of a particular chemical. Again, the quotient method of comparing measured chemical concentrations to toxicity thresholds conservatively assumes that the chronic exposures do exist. It is possible that the MFLBC indicator species move in and out of the MFLBC areas, and, therefore, their exposure is actually less than that assumed in the model.

3. Quotient Ratios and Risk Magnitudes

The quotient ratios (i.e., the magnitude of the difference between the chemical exposure at the site and the chronic toxicity threshold) are intended to correlate with the potential for, and magnitude of, risk. It is assumed that the level of risk and the magnitude of adverse effects increase as the exposure exceeds the toxicity threshold. There are, however, no ecological standards comparable to the human health standards. Considering, however, the conservatism designed into the toxicity and exposure estimates, and factoring in any available site specific information on the condition of the ecosystem, exceedances of less than a factor of 10 are not likely to relate to a high potential for risk. This is consistent with Newell's (1987) discussion on fish tissue criteria for piscivorous birds and mammals, which is referenced above. In the absence of generally accepted standards, professional judgement must be used to determine whether enough information exists to weigh the uncertainties and to make risk management decisions.

4. 4-Methylphenol Sediment Toxicity Threshold

The OEPA (1988, 1991) method of deriving water quality standards utilizes an uncertainty factor of 45 to account for the difference between acute and chronic toxicity in aquatic organisms when no chronic toxicity data are available. This was the procedure followed to derive a chronic warm water habitat criterion of 6.2 μg/kg for 4-

methylphenol. However, chronic data do exist for 4-methylphenol (Baron et al. 1984 in *AQUIRE* 1993). This data is for a chronic lowest observed effect concentration (LOEL) of 2.57 mg/L for fathead minnows. Using this chronic value and the acute toxicity value for the species provided in OEPA (1988), an acute-chronic ratio of 11.1 is calculated. Use of 11.1 instead of 45 would increase the water quality standard aproximately four-fold.

J. Recommendations

1. Indiana Bat Habitat Confirmation

Because of the inadequate resolution of available habitat data, and concerns for potentially toxic exposures for this species through the food chain, it is recommended that a field assessment of the habitat suitability of the MFLBC for the Indiana bat be conducted. This habitat information could then be used to determine if this species will play a role in any future risk-based decisions concerning the MFLBC.

2. Additional Receptor Species Habitat Confirmation

In light of the results of this ecological risk assessment, and in response to the Biological Technical Assistance Group's comments on the April 5, 1991 draft Endangerment Assessment, it is recommended <u>not</u> to conduct a field assessment of the habitat suitability for any of the other terrestrial/avian receptor species (beyond the Indiana bat) because, even under the assumptions that they reside in the MFLBC area and are chronically exposed to mirex, there is negligible potential for risk. Therefore, such a field assessment is not warranted.

X. ON-SITE ECOLOGICAL ASSESSMENT

This section evaluates potential ecological impacts associated with chemicals that have been measured on-site[8] and in the adjacent off-site areas of the Nease Chemical site (Site). Potential ecological impacts associated with these areas are evaluated separately from those potentially associated with the Middle Fork of Little Beaver Creek (MFLBC) to reflect the differences in habitat type and quality, and, consequently, exposure potentials of the two areas. This assessment follows the general approaches outlined for the MFLBC assessment, except that exposures and risks are evaluated qualitatively. A quantitative evaluation was not considered warranted, given the limited habitat quality of the Site (relative to the MFLBC), and the relatively localized nature of chemical distribution.

A. Site Description and Receptor Characterization

The Site is located in a mixed industrial-residential area, northwest of the town of Salem, Ohio. The former facility is bordered to the south and west by small manufacturing companies and private residences, to the east by the Crane-Deming Company, and to the north by a field and wooded area. Railroad tracks bisect the Site in a southeasterly direction. An inactive municipal landfill and the Salem wastewater treatment plant (WWTP) occur east and southeast of the Site, respectively, and are both adjacent to the MFLBC.

Habitat on site and in the adjacent off-site areas reflects the relatively developed nature of the Site and surrounding area, and consists principally of (mowed and unmowed) grassed uplands interspersed with successional forbs and shrubs. A few small wooded areas are scattered throughout, with the largest wooded area located north-northeast of the Site, and north of the Crane-Deming property.

Seasonal aquatic habitat is provided by several intermittent streams (including Feeder Creek and its tributaries) and ditches which drain the Site to the north, east, and south. Small pockets of palustrine emergent wetlands add to the seasonal aquatic habitat in areas where drainage has been restricted (i.e., along the railroad tracks), and/or where surficial aquifers discharge to the surface. Palustrine emergent wetlands occur on-site near the railroad tracks east of Pond 2, north and west of Pond 7, and in adjacent off-site areas west

[8] On site is defined as those areas within the Ruetgers-Nease property boundary.

of the Crane-Deming Company building and directly east of Allen Road. In addition, Slanker Pond, Feeder Pond, and a lagoon provide palustrine open water habitat north of the Site. Figure 3-2 of the remedial investigation (RI) report depicts the locations of these wetlands on and near the Site.

Wildlife species most likely to use the area are those adapted to developed/urban or field habitats. Mammalian species of the Site and surrounding area likely include shrew, opossum, mole, cottontail rabbit, and a variety of rodent species. Larger mammals, primarily fox and deer, could forage occasionally in the Site area, although access to much of the on-site area is restricted by a high fence. Bird species of the Site and surrounding area likely include urban-adapted species such as pigeon, mourning dove, crow, starling, sparrows, robin, mockingbird, and a variety of other passerine species. Quail, woodpeckers, and raptors likely occur in the off-site areas.

By far, the greatest wildlife use is expected to occur in the field and wooded areas north of the Site, where good cover and food sources exist. Wildlife use of the on-site area and adjacent industrial and residential areas is likely limited due to comparatively poorer quality cover and forage. The intermittent drainages and wetlands on-site and in adjacent off-site areas, however, probably provide seasonal habitat for aquatic life, which would consist primarily of insects and amphibian larvae. Aquatic predators of these species (e.g., rail, heron) could occasionally forage in these seasonal habitats. Frequent use is considered unlikely, however, given the small size and isolated nature of these habitats and the availability of aquatic habitat of much higher quality in the nearby MFLBC, its tributaries, and their associated riparian zone.

B. Potential Exposures and Assessment of Risk

Ecological receptors at the Site could be exposed to chemicals in surface soils, surface water, and sediment via the same pathways as discussed for receptors living or foraging in the MFLBC. Overall, however, potential exposures for most receptors are likely to be significantly less than those for the MFLBC because receptor use of the on-site area is likely to be limited by the absence of certain habitats (e.g., fish or piscivore habitat) and the relatively poor quality of the existing on-site habitat (i.e., grassy areas providing little cover or forage). For example, it is considered highly unlikely that predators such as Northern harrier or red fox would forage preferentially in the on-site area, given the presence of higher quality prey habitat (and probable higher prey density) in the fields located north of the Site. Similarly, Virginia rail or sora are unlikely to forage in the small, seasonal drainages and wetlands located on the Site, given the availability of much higher quality

aquatic habitat in the nearby MFLBC and its tributaries. Admittedly, such exposures could occur intermittently, but these infrequent exposures are not likely to result in adverse effects.

The Site, however, does pose a threat to resident species that are relatively sedentary and/or that have small home ranges and, consequently, that could be exposed more frequently to elevated concentrations of chemicals in soils, sediments, and surface waters. These receptors at risk include terrestrial species, such as earthworms and other soil-dwelling invertebrates, and small mammals and birds that may feed on soil invertebrates or live in the soil, and aquatic species, such as insect and amphibian larvae.

1. Terrestrial Species

It is considered likely that earthworms and other soil invertebrates have been affected by some of the chemicals detected in the soils at the Site. Although chemical-specific toxicity information for soil invertebrates is generally lacking for the chemicals detected at the Site, toxicity data on a number of other chemicals suggest that the maximum on-site soil concentrations of mirex (> 2,000 mg/kg) and total volatile and semi-volatile chemicals (> 40,000 and 1,000 mg/kg, respectively at some locations) are likely to be toxic to some species. For example, Neuhauser et al. (1985) reported median lethal concentration (LC_{50}) values for earthworms in the range of 30 to greater than 4,000 mg/kg for a variety of semivolatile chemicals. If such toxicity is occurring, it is likely to be limited spatially given that the maximum chemical concentrations at the Site are relatively localized. For example, maximum mirex soil concentrations are limited to areas near and beneath the on-site buildings, Pond 1, and Pond 2, with concentrations in most of the remaining areas of approximately 1 mg/kg (see Figure 4-X of the RI Report).

Small mammals that forage exclusively or predominantly on-site may be at risk from exposure to elevated chemical concentrations. Insectivorous species such as shrews may be at risk from exposures to chemicals that have accumulated in prey (i.e., soil invertebrates), although overall exposure would be mitigated somewhat by the likely absence of such prey in the high chemical concentration areas. In addition, soil-dwelling mammals could be exposed via direct ingestion of soil and inhalation of soil vapors (in areas with high volatile chemical concentrations). Soil ingestion alone could

result in chronic toxicity in small mammals exposed to the average, site-wide mirex concentration of approximately 74 mg/kg, although acute toxicity is not likely.[9]

2. Aquatic Species

Aquatic insects and amphibian larvae present seasonally in the on-site drainages potentially could be affected by the elevated chemical concentrations present locally in these systems. The areas of greatest potential exposure are the tributaries of the Feeder Creek system where maximum surface water and sediment concentrations of mirex, plus volatile and semivolatile chemicals have been detected. The mirex surface water concentration at station FC-3, located southeast of Pond 3, was reported to be 0.36 ug/L, which exceeds the lowest-observed-effect concentration (LOEC) of 0.1 ug/L reported for crayfish, the most sensitive aquatic species studied to date. Although it is not known if aquatic insects or amphibian larvae are as sensitive to mirex as crayfish, surface water exposures in combination with exposures to elevated mirex concentrations in the sediment at this location ($\sim$ 118 mg/kg) could result in a toxic response.

C. Conclusions

It is likely that chemicals present on-site at the Nease Chemical Site may impact select groups of ecological receptors. These ecological impacts are expected to be localized given the chemical distribution at the Site and limited to species such as terrestrial or aquatic invertebrates, amphibians, small mammals, and birds that are relatively sedentary and/or have limited home ranges. More wide-ranging species, such as those likely to frequent the MFLBC and nearby habitats, are unlikely to be at risk due to chemical exposures from the Site.

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[9] For example, soil ingestion by meadow voles (used here as a surrogate small mammal) could exceed the toxicity criterion of 0.1 mg/kg-day, assuming a vole ingests 0.00018 kg soil/day (Beyer 1992) and weighs 50 g (Burt and Grossenheider 1976): [74 mg mirex/kg soil * 0.00018 kg soil/day] / 0.05 kg bw = 0.27 mg/kg-day. This is below acutely toxic concentrations reported to range from 5 to > 500 mg/kg-day (Eisler 1985).

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TABLE 1

CHEMICALS DETECTED IN VARIOUS SAMPLED MEDIA

	CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL
	VOLATILE COMPOUNDS										
1	1,1-Dichloroethene	x									
2	1,1,1-Trichloroethane		x				x				
3	1,1,2-Trichloroethane	x	x	x	x	x					
4	1,1,2,2-Tetrachloroethane	x	x	x	x	x					
5	1,2-Dichloroethane	x	x	x	x	x				x	
6	1,2-Dichloroethene (total)	x	x	x	x	x					
7	1,2-Dichloropropane		x	x		x				x	
8	2-Butanone		x	x					x	x	
9	Acetone	x		x	x				x	x	
10	Benzene	x	x	x	x	x			x		
11	Bromoform	x		x	x						
12	Carbon Disulfide	x	x	x							
13	Carbon Tetrachloride			x	x		x				
14	Chlorobenzene	x	x	x	x	x	x				
15	Chloroethane	x									
16	Chloroform	x	x	x	x	x					
17	Chloromethane	x		x	x			x			
18	Dibromochloromethane			x	x						
19	Ethylbenzene	x		x	x		x		x		
20	Methylene Chloride	x		x	x	x			x		
21	Styrene	x		x			x				
22	Tetrachloroethene	x	x	x	x	x			x		
23	Toluene	x	x	x	x	x			x		
24	Trichloroethene	x	x	x	x	x	x				
25	Vinyl Chloride	x	x								

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26	Xylene (total)		x	x	x	x	x		x		
	SEMIVOLATILE COMPOUNDS										
27	1,2-Dichlorobenzene	x	x	x	x	x					
28	1,2,4-Trichlorobenzene	x		x		x					
29	1,3-Dichlorobenzene	x		x							
30	1,4-Dichlorobenzene	x	x	x		x					
31	2-Chlorophenol	x		x							
32	2-Methylnaphthalene	x	x	x		x				x	
33	2-Methylphenol	x									
34	2-Nitroaniline	x									
35	2-Nitrophenol	x			x						
36	2,4-Dichlorophenol	x	x	x	x	x					
37	2,4-Dimethylphenol	x		x							
38	2,4,6-Trichlorophenol	x	x								
39	2,6-Dinitrotoluene	x									
40	3-Nitroaniline	x									
41	4-Chloro-3-Methylphenol	x		x					x		
42	4-Methylphenol	x		x		x				x	
43	Acenaphthene	x	x			x				x	
44	Acenaphthylene		x			x					
45	Anthracene		x			x				x	
46	Benzoic Acid			x	x	x			x	x	
47	Benzo(a)Anthracene		x	x		x				x	
48	Benzo(a)Pyrene		x	x		x				x	
49	Benzo(b)Fluoranthene		x	x		x				x	

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	CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL
50	Benzo(g,h,i)Perylene			x		x				x	
51	Benzo(k)Fluoranthene		x	x		x				x	
52	Benzyl Alcohol			x	x						
53	bis(2-Chloroethoxy)Methane	x									
54	bis(2-Chloroethyl)Ether	x									
55	bis(2-Ethylhexyl)Phthalate	x		x	x	x	x	x		x	
56	Butylbenzylphthalate	x	x	x		x			x		
57	Carbazole	x									
58	Chrysene		x	x		x				x	
59	Dibenzofuran		x	x		x				x	
60	Diethylphthalate	x	x	x		x					
61	Dimethylphthalate			x					x		
62	Di-n-Butylphthalate	x		x		x			x	x	
63	Di-n-Octylphthalate	x									
64	Diphenylsulfone	x	x	x	x	x		x		x	
65	Fluoranthene		x	x		x				x	
66	Fluorene		x			x				x	
67	Hexachlorobenzene	x	x	x							
68	Hexachlorobutadiene	x	x	x							
69	Hexachlorocyclopentadiene			x							
70	Hexachloroethane	x	x	x	x						
71	Indeno(1,2,3-cd)Pyrene		x			x				x	
72	Naphthalene	x	x	x		x				x	
73	Nitrobenzene	x									
74	N-Nitrosodiphenylamine			x		x	x		x		
75	N-Nitroso-di-n-Propylamine	x									

TABLE 1

CHEMICALS DETECTED IN VARIOUS SAMPLED MEDIA

	CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL
76	Pentachlorophenol			x							
77	Phenanthrene	x	x	x		x				x	
78	Phenol	x	x	x					x	x	
79	Pyrene	x	x	x		x				x	
	PESTICIDES										
80	4,4'-DDD	x	x			x					
81	4,4'-DDE	x				x			x	x	
82	4,4'-DDT	x		x		x				x	
83	Aldrin	x							x		
84	alpha-BHC	x									
85	alpha-Chlordane	x							x		
86	beta-BHC	x		x					x		
87	delta-BHC			x							
88	Dieldrin	x	x	x		x					
89	Endosulfan I	x							x		
90	Endosulfan II	x									
91	Endosulfan sulfate	x									
92	Endrin	x		x					x		
93	Endrin aldehyde	x									
94	gamma-BHC (Lindane)	x							x		
95	gamma-Chlordane	x									
96	Heptachlor	x								x	
97	Methoxychlor	x	x	x	x						

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CHEMICALS DETECTED IN VARIOUS SAMPLED MEDIA

	CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL
	OTHER										
98	Kepone	x		x	x	x					
99	Mirex	x	x	x	x	x	x		x	x	x
100	Photomirex	x	x	x	x	x	x		x	x	x
	DIOXINS AND FURANS										
101	1,2,3,4,6,7,8-HpCDD	x		x							
102	1,2,3,4,6,7,8-HpCDF	x									
103	1,2,3,4,7,8-HxCDF	x									
104	1,2,3,4,7,8,9-HpCDF	x									
105	1,2,3,6,7,8-HxDCF	x									
106	1,2,3,7,8-PeCDF	x		x							
107	2,3,4,6,7,8-HxCDF	x									
108	2,3,4,7,8-PeCDF	x									
109	2,3,7,8-TCDD	x									
110	2,3,7,8-TCDF	x		x							
111	OCDD	x		x							
112	OCDF	x		x							
113	Total HpCDDs	x		x							
114	Total HpCDFs	x		x							
115	Total HxCDDs	x									
116	Total HxCDFs	x		x							
117	Total PeCDDS	x									
118	Total PeCDFs	x		x							
119	Total TCDD	x									
120	Total TCDFs	x		x							

TABLE 1

CHEMICALS DETECTED IN VARIOUS SAMPLED MEDIA

	CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL
	INORGANICS										
121	Aluminum	x		x		x					
122	Antimony	x		x		x					
123	Arsenic	x		x		x					
124	Barium	x		x		x					
125	Beryllium	x		x		x					
126	Cadmium	x		x		x					
127	Calcium	x		x		x					
128	Chromium	x		x		x					
129	Cobalt	x		x		x					
130	Copper	x		x		x					
131	Iron	x		x		x					
132	Lead	x		x		x					
133	Magnesium	x		x		x					
134	Manganese	x		x		x					
135	Mercury	x		x		x					
136	Nickel	x		x		x					
137	Potassium	x		x		x					
138	Selenium			x		x					
139	Silver	x		x		x					
140	Sodium	x		x		x					
141	Thallium	x		x							
142	Vanadium	x		x		x					
143	Zinc	x		x		x					
144	Cyanide	x		x		x					

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CHEMICALS DETECTED IN VARIOUS SAMPLED MEDIA

CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL

NOTE: "x" indicates chemical detected in at least one sample in the medium; no entry indicates chemical not detected in medium.

TABLE 2

CHEMICALS DETECTED IN ON-SITE TEST PIT SOIL

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Methylene chloride	4	81	3	990	2	210
Acetone	10	80	10	1200	11	430
1,2-Dichloroethene (total)	6	81	5.4	600	1	350
Chloroform	1	81	5.4	600	ND	12
1,2-Dichloroethane	11	81	5.4	600	3	990
2-Butanone	11	76	10.8	1200	2	78
1,1,2,2-Tetrachloroethane	25	81	5.4	600	1	9000
1,2-Dichloropropane	16	81	5.4	600	1	73
Trichloroethene	24	81	5.4	600	1	2200
1,1,2-Trichloroethane	5	81	5.4	600	3	180
Benzene	10	81	5.4	600	2	900
Bromoform	1	81	5.4	600	ND	35
Tetrachloroethene	37	81	5.4	600	1	13000
Toluene	8	81	5.4	600	1	290
Chlorobenzene	13	81	5.4	600	1	120
Ethylbenzene	3	81	5.4	600	1	570
Xylene (total)	1	81	5.4	600	ND	13
SEMI-VOLATILE COMPOUNDS						
Phenol	4	79	340	30000	68	220
2-Chlorophenol	2	79	340	30000	50	99
1,3-Dichlorobenzene	1	79	340	30000	ND	69
1,4-Dichlorobenzene	4	79	340	30000	46	3000
Benzyl alcohol	1	79	340	30000	ND	80
1,2-Dichlorobenzene	16	79	340	30000	47	290000
4-Methylphenol	1	79	340	30000	ND	83
Hexachloroethane	6	79	340	30000	150	400
2,4-Dimethylphenol	1	79	340	30000	ND	390
Benzoic acid	1	79	1700	150000	ND	490
2,4-Dichlorophenol	3	79	340	30000	43	430
1,2,4-Trichlorobenzene	1	79	340	30000	ND	140
Naphthalene	7	79	340	30000	42	340
Hexachlorobutadiene	4	79	340	30000	80	1400
4-Chloro-3-methylphenol	1	79	340	30000	ND	38
2-Methylnaphthalene	7	79	340	30000	44	400
Hexachlorocyclopentadiene	3	79	340	30000	57	250
Dimethylphthalate	3	79	340	30000	99	350
Dibenzofuran	1	79	340	30000	ND	210
Diethylphthalate	5	80	340	30000	55	1100
N-Nitrosodiphenylamine	7	80	340	30000	53	440
Hexachlorobenzene	19	80	340	30000	39	2900
Pentachlorophenol	2	79	340	30000	360	8600
Phenanthrene	10	79	340	30000	38	410
Di-n-butylphthalate	2	80	340	30000	41	1100
Fluoranthene	5	79	340	30000	41	110

TABLE 2

CHEMICALS DETECTED IN ON-SITE TEST PIT SOIL

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
Pyrene	3	79	340	30000	76	240
Butylbenzylphthalate	1	80	340	30000	ND	1300
Benzo(a)anthracene	1	79	340	30000	ND	60
Chrysene	2	79	340	30000	71	300
Bis(2-ethylhexyl)phthalate	30	81	340	3861	43	940
Benzo(b)fluoranthene	5	79	340	30000	42	340
Benzo(k)fluoranthene	5	79	340	30000	42	390
Benzo(a)pyrene	2	79	340	30000	57	190
Benzo(g,h,i)perylene	1	79	340	30000	ND	38
Diphenylsulfone	32	80	340	30000	47	540000
PESTICIDES						
Beta-BHC	1	76	4	480	ND	180
Delta-BHC	1	76	4	480	ND	76
Dieldrin	1	79	8	960	ND	13
Endrin	1	78	8	960	ND	16
4,4'-DDT	1	78	8	960	ND	100
Methoxychlor	1	70	8	960	ND	380
OTHER COMPOUNDS						
Kepone	6	75	72	2000000	74.5	85.8
Photomirex	25	80	22	600000	0.279	559
Mirex	78	81	20	540000	2.1	2080000
INORGANIC COMPOUNDS						
Aluminum	19	19	3	3	6810	20600
Arsenic	19	19	0.4	0.4	9	23.1
Barium	19	19	0.2	0.2	23.5	116
Beryllium	19	19	0.2	0.2	0.42	1.1
Cadmium	4	19	0.6	1.8	1	1.6
Calcium	14	19	2.8	2.8	972	103000
Chromium	19	19	0.6	0.6	11.3	22.5
Cobalt	18	19	0.6	4.3	7.2	14.5
Copper	19	19	0.6	0.6	15.6	37.6
Iron	19	19	0.6	0.6	19600	43300
Lead	19	19	0.4	0.4	11.5	24.8
Magnesium	14	19	4.6	4.6	1990	6860
Manganese	19	19	0.2	0.2	230	1150
Mercury	3	19	0.1	0.1	0.1	0.2
Nickel	12	19	4.8	4.8	13.1	34.9
Potassium	19	19	85.6	85.6	493	2180
Selenium	3	19	0.4	0.4	0.43	1.3
Silver	1	19	0.4	0.57	ND	0.7
Sodium	4	19	3.6	155	52.6	113
Thallium	2	19	0.6	0.6	0.77	1

TABLE 2

CHEMICALS DETECTED IN ON-SITE TEST PIT SOIL

CHEMICAL	Detection Frequency: Number of Detects	Detection Frequency: Number of Samples	Range of Reported Quantitation Limits (ug/kg): Minimum	Range of Reported Quantitation Limits (ug/kg): Maximum	Range of Detected Concentrations (ug/kg): Minimum	Range of Detected Concentrations (ug/kg): Maximum
Vanadium	19	19	0.6	0.6	13.3	42.6
Zinc	12	19	1.2	1.2	56.2	138
Cyanide	1	19	0.25	0.25	ND	0.4
DIOXINS AND FURANS						
Total HpCDDs	1	23	0.0052	0.075	ND	0.095
1,2,3,4,6,7,8-HpCDD	1	23	0.0052	0.075	ND	0.038
OCDD	7	23	0.015	0.13	0.082	0.37
Total TCDFs	3	23	0.0028	0.026	0.016	0.53
2,3,7,8-TCDFs	1	23	0.0028	0.026	ND	0.15
Total PeCDFs	1	23	0.0027	0.023	ND	0.43
1,2,3,7,8-PeCDF	1	23	0.0027	0.023	ND	0.27
Total HxCDFs	1	23	0.0033	0.071	ND	0.13
Total HpCDFs	1	23	0.0049	0.061	ND	0.26
OCDF	1	23	0.018	0.16	ND	0.28

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 3

CHEMICALS DETECTED IN ON-SITE SOIL (POND)

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Chloromethane	2	66	11	110000	450	1500
Acetone	13	64	11	110000	78	98000
Carbon Disulfide	1	65	5.5	75000	ND	16000
1,2-Dichloroethene (total)	3	64	5.5	55000	3	10
Chloroform	15	69	5.5	55000	2	45000
1,2-Dicholoethane	32	69	5.5	55000	3	250000
Carbon Tetrachloride	8	66	5.5	55000	1000	70000
1,1,2,2-Tetrachloroethane	37	75	5.5	600000	1	7200000
1,2-Dichloropropane	2	64	5.5	55000	2	72
Trichloroethene	55	74	5.5	600000	2	2200000
Dibromochloromethane	1	65	5.5	55000	ND	8700
Benzene	54	75	5.5	600000	1	4700000
Bromoform	9	66	5.5	55000	370	34000
Tetrachloroethene	54	76	5	600000	3	38000000
Toluene	39	75	5.5	600000	1	550000
Chlorobenzene	35	74	5.5	75000	4	300000
Ethylbenzene	11	67	5.5	55000	1	57000
Styrene	5	64	5.5	55000	11	420000
Xylene (total)	17	69	5.5	55000	6	250000
SEMI-VOLATILE COMPOUNDS						
Phenol	13	75	369.6	808500	75	270
1,3-Dichlorobenzene	1	75	369.6	808500	ND	1200
1,4-Dichlorobenzene	23	75	369.6	808500	59	27000
1,2-Dichlorobenzene	36	74	369.6	808500	41	2700000
Hexachloroethane	17	75	369.6	808500	320	330000
Benzoic acid	24	76	1914.0	4042500	360	570000
2,4-Dichlorophenol	3	75	369.6	808500	370	3900
1,2,4-Trichlorobenzene	6	75	369.6	808500	810	3700
Naphthalene	9	75	369.6	808500	95	3200
Hexachlorobutadiene	10	75	369.6	808500	810	56000
2-Methylnaphthalene	2	75	369.6	808500	54	180
Diethylphthalate	4	75	369.6	808500	160	19000
N-Nitrosodiphenylamine	13	75	369.6	808500	78	13000
Hexachlorobenzene	22	75	369.6	808500	160	100000
Phenanthrene	3	75	369.6	808500	52	140
Di-n-butylphthalate	1	75	369.6	808500	ND	42
Bis(2-ethylhexyl)phthalate	24	75	369.6	808500	100	4000
Benzo(g,h,i)perylene	3	75	369.6	808500	41	80
Diphenylsulfone	59	76	369.6	808500	49	7400000
PESTICIDES						
Dieldrin	1	69	18.24	40000	ND	3000
4,4'-DDT	1	70	18.24	40000	ND	96

TABLE 3

CHEMICALS DETECTED IN ON-SITE SOIL (POND)

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
Methoxychlor	32	68	91.2	200000	36	280000
OTHER						
Kepone	3	63	66.6	312800	63.5	761
Photomirex	7	75	19.99	244800	0.728	33.4
Mirex	66	76	18.13	222000	1.06	938000
INORGANICS						
Aluminum	18	18	3000	3000	755000	17200000
Antimony	5	18	3800	7600	6900	36700
Arsenic	18	18	400	400	1400	17800
Barium	18	18	200	200	3200	115000
Beryllium	12	18	200	200	330	940
Cadmium	1	18	600	1200	ND	890
Calcium	18	18	2800	2800	1350000	287000000
Chromium	18	18	600	600	2500	47500
Cobalt	12	18	600	2800	1800	11700
Copper	15	18	600	17200	5100	38400
Iron	18	18	600	1370000	1370000	33800000
Lead	18	18	400	3500	3500	26900
Magnesium	18	18	4600	213000	213000	6270000
Manganese	18	18	200	33400	33400	669000
Mercury	2	18	100	110	110	300
Nickel	13	18	4800	9600	8000	29800
Potassium	14	18	85600	171200	376000	2500000
Selenium	4	18	400	400	510	2100
Sodium	18	18	3600	3600	54000	1060000
Thallium	1	18	600	600	ND	9800
Vanadium	17	18	600	2600	2700	31400
Zinc	18	18	1200	1200	4300	611000
Cyanide	2	18	250	250	890	1400
DIOXINS AND FURANS						
OCDD	9	24	0.024	1.3	0.11	3
Total TCDFs	1	24	0.0039	0.15	ND	0.014
2,3,7,8-TCDF	1	24	0.0039	0.15	ND	0.071

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 4

CHEMICALS DETECTED IN GROUND WATER

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/l)		Range of Detected Concentrations (ug/l)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
Chloromethane	12	152	10	10000	1	47
Vinyl chloride	26	152	10	10000	2	1700
Chloroethane	1	152	10	10000	ND	4
Methylene Chloride	1	152	10	10000	ND	42
Acetone	2	152	10	10000	7	530
Carbon disulfide	1	152	10	10000	ND	3
1,1-Dichloroethene	14	152	10	10000	1	130
1,2-Dichloroethene (total)	51	152	10	10000	2	19000
Chloroform	14	152	10	10000	1	1200
1,2-Dichloroethane	43	152	10	10000	2	23000
1,1,2,2-Tetrachloroethane	20	152	10	10000	9	60000
Trichloroethene	41	152	10	10000	2	20000
1,1,2-Trichloroethane	3	152	10	10000	3	110
Benzene	47	152	10	10000	1	45000
Bromoform	1	152	10	10000	ND	680
Tetrachloroethene	32	152	10	10000	1	100000
Toluene	27	152	10	10000	1	23000
Chlorobenzene	40	152	10	10000	1	4700
Ethylbenzene	5	152	10	10000	3	1200
Styrene	3	152	10	10000	1	6
SEMI-VOLATILE COMPOUNDS						
Phenol	53	143	10	5000	1	400
Bis(2-chloroethyl)ether	2	139	10	5000	1	1
2-Chlorophenol	11	141	10	5000	1	18
1,3-Dichlorobenzene	11	140	10	5000	1	6
1,4-Dichlorobenzene	35	143	10	5000	1	300
1,2-Dichlorobenzene	44	143	10	5000	1	36000
2-Methylphenol	3	142	10	5000	3	48
4-Methylphenol	9	141	10	5000	1	42
N-Nitroso-di-n-Propylamine	1	139	10	5000	ND	1
Hexachloroethane	10	140	10	5000	15	470
Nitrobenzene	4	141	10	5000	11	120
2-Nitrophenol	2	139	10	5000	1	3
2,4-Dimethylphenol	5	140	10	5000	1	7
bis(2-Chloroethoxy)methane	1	139	10	5000	ND	2
2,4-Dichlorophenol	21	143	10	5000	1	670
1,2,4-Trichlorobenzene	6	139	10	5000	3	18
Naphthalene	14	140	10	5000	1	97
Hexachlorobutadiene	9	139	10	5000	2	110
4-Chloro-3-methylphenol	1	140	10	5000	ND	660
2-Methylnaphthalene	3	139	10	5000	1	2
2,4,6-Trichlorophenol	4	139	10	5000	1	6
2-Nitroaniline	12	142	25	12500	4	68
3-Nitroaniline	2	140	20	12500	7	14
Acenaphthene	1	139	10	5000	ND	6
2,6-Dinitrotoluene	3	141	10	5000	14	79

TABLE 4

CHEMICALS DETECTED IN GROUND WATER

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/l)		Range of Detected Concentrations (ug/l)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
Diethylphthalate	1	139	10	5000	ND	1
Hexachlorobenzene	8	139	10	5000	1	130
Phenanthrene	1	139	10	5000	ND	1
Carbazole	3	140	10	5000	1	36
Di-n-Butylphthalate	1	140	10	5000	ND	1
Pyrene	1	139	10	5000	ND	1
Bis(2-ethylhexyl)phthalate	13	147	10	5000	1	920
Di-n-Octylphthalate	1	139	10	5000	ND	6
Diphenyl sulfone	52	144	10	5000	1	14000
Butylbenzyl phthalate	6	139	10	5000	1	6
PESTICIDES						
Alpha-BHC	5	136	0.05	5	0.0015	0.042
Beta-BHC	2	143	0.05	5	0.003	0.0048
Gamma-BHC (Lindane)	2	128	0.05	5	0.023	0.13
Heptachlor	15	131	0.05	5	0.0043	0.024
Aldrin	3	138	0.05	5	0.0078	0.076
Endosulfan I	2	140	0.05	5	0.62	2.1
Dieldrin	15	145	0.1	10	0.0013	0.018
4,4'-DDE	4	143	0.1	10	0.0022	0.089
Endrin	10	144	0.1	10	0.0021	4.4
Endosulfan II	4	145	0.1	10	0.017	2.2
4,4'-DDD	7	143	0.1	10	0.0077	53
Endosulfan sulfate	2	145	0.1	10	0.024	0.036
4,4'-DDT	14	141	0.1	10	0.0028	3.2
Methoxychlor	14	146	0.5	50	0.015	2.4
Endrin aldehyde	8	147	0.1	10	0.026	14
Alpha-chlordane	6	145	0.05	5	0.0022	0.28
Gamma-chlordane	9	146	0.05	5	0.0015	0.44
OTHER COMPOUNDS						
Kepone	6	145	0.132	264	0.067	13.1
Photomirex	18	147	0.0474	94.8	0.005	4.83
Mirex	64	148	0.00544	10.88	0.001511	239.6
DIOXINS AND FURANS						
total TCDD	4	6	0.0000105	0.0000107	0.000016	0.000106
2,3,7,8-TCDD	2	5	0.0000105	0.0000109	0.0000147	0.0000167
Total PeCDDS	3	6	0.0000525	0.0000546	0.0000651	0.000257
Total HxCDDs	3	7	0.0000525	0.0000546	0.0000603	0.000187
Total HpCDDs	5	8	0.0000525	0.0000546	0.000145	0.000348
1,2,3,4,6,7,8-HpCDD	5	8	0.0000525	0.0000546	0.0000814	0.000181
OCDD	6	7	0.0000107	0.0000107	0.00012	0.000532
Total TCDFs	7	8	0.0000107	0.0000107	0.000144	0.003374
2,3,7,8-TCDF	6	8	0.0000105	0.0000107	0.0000146	0.000231

TABLE 4

CHEMICALS DETECTED IN GROUND WATER

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/l)		Range of Detected Concentrations (ug/l)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
Total PeCDFs	6	8	0.0000525	0.0000534	0.000771	0.00243
1,2,3,7,8-PeCDF	3	7	0.0000525	0.0000546	0.0000641	0.000098
2,3,4,7,8-PeCDF	6	8	0.0000525	0.0000534	0.0000659	0.000272
Total HxCDFs	6	8	0.0000525	0.0000534	0.000216	0.00129
1,2,3,4,7,8-HxCDF	6	8	0.0000525	0.0000534	0.0000944	0.000283
1,2,3,6,7,8-HxDCF	4	8	0.0000525	0.0000546	0.000114	0.00016
2,3,4,6,7,8-HxCDF	4	8	0.0000525	0.0000546	0.0000735	0.000119
Total HpCDFs	7	8	0.0000534	0.0000534	0.0000485	0.00139
1,2,3,4,6,7,8-HpCDF	7	8	0.0000534	0.0000534	0.0000479	0.000526
1,2,3,4,7,8,9-HpCDF	4	8	0.0000525	0.0000546	0.0000605	0.000112
OCDF	6	8	0.000106	0.000107	0.000158	0.000486
INORGANIC COMPOUNDS						
Aluminum	7	8	19.3	44.2	476.6	754230
Antimony	1	8	2.6	6.4	ND	5.9
Arsenic	8	8	1	1.6	5.6	70.4
Barium	7	8	0.4	30.5	32.39	239.83
Beryllium	7	8	0.2	3	0.69	78
Cadmium	6	7	1.7	8.5	4.33	123
Calcium	8	8	3.7	10.7	182270	775810
Chromium	6	8	2.9	88.65	5.22	171.2
Cobalt	7	8	2.4	13.5	4.22	994.4
Copper	6	8	1.6	21.25	244.21	2141.4
Iron	8	8	4.8	5.7	14999	716630
Lead	5	8	0.9	3.5	12.7	756
Magnesium	8	8	37.2	43.7	30996	313990
Manganese	8	8	0.4	1.1	426.06	101960
Mercury	6	8	0.2	0.2	0.26	1.77
Nickel	7	8	4.6	94.4	29.87	2265.1
Potassium	7	7	339.2	1700	2552.7	17033
Silver	4	8	2.4	2.4	11.49	24.69
Sodium	8	8	14.7	138.1	110780	1736700
Thallium	2	8	1.2	1.2	1.9	5.4
Vanadium	5	8	2	14	93.2	635.85
Zinc	8	8	2.3	3.7	162.02	7949.9
Cyanide	3	8	10	10	15	25.3

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 5

CHEMICALS DETECTED IN AIR

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (mg/m^3)		Range of Detected Concentrations (mg/m^3)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
1,1,1-Trichloroethane	3	8			2.89E-05	7.44E-05
Carbon Tetrachloride	8	8			9.70E-06	1.67E-05
Trichloroethene	2	8			1.23E-05	1.52E-05
Chlorobenzene	2	8			8.60E-06	1.56E-05
Ethylbenzene	2	8			7.10E-06	1.43E-05
Xylene (total)	6	8			7.50E-06	5.71E-05
Styrene	1	8			ND	8.80E-06
SEMI-VOLATILE COMPOUNDS						
N-Nitrosodiphenylamine	1	6			ND	2.20E-03
Bis-(2-Ethylhexyl)Phthalate	2	6			4.10E-03	2.55E-02
OTHER						
Photomirex	7	7			8.70E-07	5.63E-06
Mirex	7	7			4.88E-06	4.70E-05

Quantitation limits were not reported for air samples.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 6

CHEMICALS DETECTED IN ON-SITE SEDIMENTS

CHEMICAL	Detection Frequency: Number of Detects	Detection Frequency: Number of Samples	Range of Reported Quantitation Limits (ug/kg): Minimum	Range of Reported Quantitation Limits (ug/kg): Maximum	Range of Detected Concentrations (ug/kg): Minimum	Range of Detected Concentrations (ug/kg): Maximum
VOLATILE COMPOUNDS						
Vinyl chloride	1	7	14	38	ND	14
Carbon disulfide	1	7	14	38	ND	4
1,2-Dichloroethene (total)	4	7	14	100	22	280
Chloroform	2	7	14	38	2	16
1,2-Dichloroethane	4	7	14	100	5	640
2-Butanone	1	7	14	38	ND	4
1,1,1-Trichloroethane	2	7	14	38	3	5
1,1,2,2-Tetrachloroethane	4	7	14	100	3	370
1,2-Dichloropropane	1	7	14	38	ND	3
Trichloroethene	5	7	14	100	2	420
1,1,2-Trichloroethane	2	7	14	38	5	10
Benzene	3	7	14	100	2	59
Tetrachloroethene	3	7	14	100	12	1900
Toluene	2	7	14	38	2	8
Chlorobenzene	2	7	14	100	12	20
Xylene (total)	1	7	14	38	ND	4
SEMI-VOLATILE COMPOUNDS						
Phenol	2	7	450	1300	43	60
1,4-Dichlorobenzene	2	7	450	1300	96	260
1,2-Dichlorobenzene	6	7	450	1300	210	6800
Hexachloroethane	2	7	450	1300	98	220
2,4-Dichlorophenol	2	7	450	1300	51	420
Naphthalene	3	7	450	1300	67	260
Hexachlorobutadiene	2	7	450	1300	160	230
2-Methylnaphthalene	3	7	450	1300	110	460
2,4,6-Trichlorophenol	1	7	450	1300	ND	95
Acenaphthylene	1	7	450	1300	ND	48
Acenaphthene	1	7	450	1300	ND	33
Dibenzofuran	3	7	450	1300	46	110
Diethylphthalate	1	7	450	1300	ND	1300
Fluorene	1	7	450	1300	ND	45
Hexachlorobenzene	3	7	450	1300	47	3000
Phenanthrene	5	7	450	1300	26	530
Anthracene	2	7	450	1300	38	110
Fluoranthene	3	7	450	1300	81	730
Pyrene	3	7	450	1300	71	920
Butylbenzylphthalate	1	7	450	1300	ND	160
Benzo(a)anthracene	5	7	450	1300	47	410
Chrysene	5	7	450	1300	56	510
Benzo(b)fluoranthene	4	7	450	1300	40	470
Benzo(k)fluoranthene	4	7	450	1300	44	370
Benzo(a)pyrene	3	7	450	1300	38	310
Indeno(1,2,3-cd)pyrene	1	7	450	1300	ND	24
Diphenylsulfone	3	7	450	1300	1100	10000

TABLE 6

CHEMICALS DETECTED IN ON-SITE SEDIMENTS

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
PESTICIDES						
Dieldrin	2	7	4.5	50	4.7	13
4,4'-DDD	1	7	4.5	50	ND	11
Methoxychlor	3	7	23	260	30	1600
OTHER COMPOUNDS						
Photomirex	5	7	29	88.9	36.6	530
Mirex	7	7	26	81	136	1100

Minimum detected concentration could be less than minimum qunatitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 7

CHEMICALS DETECTED IN ON-SITE SURFACE WATER

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/l)		Range of Detected Concentrations (ug/l)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Chloromethane	1	7	10	50	ND	3
Acetone	1	7	10	50	ND	170
1,2-Dichloroethene (total)	2	7	5	25	8	14
Chloroform	2	7	5	25	1	3
1,2-Dichloroethane	2	7	5	25	69	100
Carbon Tetrachloride	1	7	5	25	ND	2
1,1,2,2-Tetrachloroethane	2	7	5	25	140	470
Trichloroethene	2	7	5	25	82	94
Dibromochloromethane	1	7	5	25	ND	2
1,1,2-Trichloroethane	2	7	5	25	4	5
Benzene	2	7	5	25	38	140
Bromoform	1	7	5	25	ND	6
Tetrachloroethene	3	7	5	25	10	340
Toluene	1	7	5	25	ND	25
Chlorobenzene	2	7	5	25	1	13
Ethylbenzene	1	7	5	25	ND	2
Xylene (total)	1	7	5	25	ND	6
SEMI-VOLATILE COMPOUNDS						
Benzyl Alcohol	2	8	2	30	7	11
1,2-Dichlorobenzene	3	8	2	30	13	180
Hexachloroethane	1	8	2	30	ND	5
2-Nitrophenol	1	8	2	30	ND	1
Benzoic Acid	1	8	10	150	ND	310
2,4-Dichlorophenol	1	8	2	30	ND	4
bis(2-Ethylhexyl)Phthalate	1	8	2	30	ND	0.6
Diphenylsulfone	3	8	2	30	110	450
PESTICIDES						
Methoxychlor	1	8	0.5	0.5	ND	0.67
OTHER						
Kepone	1	8	0.132	0.132	ND	0.292
Photomirex	1	8	0.0474	0.0474	ND	0.0151
Mirex	3	8	0.00544	0.00544	0.0304	0.362

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 8

CHEMICALS DETECTED IN OFF-SITE SOIL BORINGS

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
1,2-Dichloroethane	2	3	5.9	6	5	6
1,2-Dichloropropane	3	3	5.9	6	27	90
Xylene (total)	1	3	5.9	6	ND	6
SEMI-VOLATILE COMPOUNDS						
1,4-Dichlorobenzene	2	22	333	1500	76	88
4-Methylphenol	3	22	333	1500	40	270
Benzoic acid	1	22	333	1500	ND	450
Naphthalene	5	22	333	1500	34	270
2-Methylnaphthalene	5	22	333	1500	56	440
Acenaphthylene	1	22	333	1500	ND	41
Acenaphthene	1	22	333	1500	ND	49
Dibenzofuran	3	22	333	1500	47	100
Diethylphthalate	5	22	333	1500	43	70
Fluorene	1	22	333	1500	ND	64
N-Nitrosodiphenylamine	2	23	333	1500	49	200
Phenanthrene	5	22	333	1500	72	1400
Anthracene	1	22	333	1500	ND	400
Di-n-butylphthalate	10	22	333	1500	46	310
Fluoranthene	6	22	333	1500	53	2100
Pyrene	5	22	333	1500	41	2000
Butylbenzylphthalate	7	22	333	1500	170	5400
Benzo(a)anthracene	3	22	333	1500	64	1400
Chrysene	3	22	333	1500	60	1300
Bis(2-ethylhexyl)phthalate	12	23	48	570	110	4100
Benzo(b)fluoranthene	5	22	333	1500	50	2400
Benzo(k)fluoranthene	5	22	333	1500	50	2400
Benzo(a)pyrene	3	22	333	1500	53	1200
Indeno(1,2,3-cd)pyrene	2	22	333	1500	47	420
Benzo(g,h,i)perylene	2	22	333	1500	43	420
PESTICIDES						
Dieldrin	2	23	16	49	6.2	60
4,4'-DDD	4	23	16	49	6.4	111
4,4'-DDE	1	23	16	49	ND	9.6
4,4'-DDT	2	23	16	49	8.2	110
OTHER COMPOUNDS						
Photomirex	1	22	22	32	ND	15.4
Mirex	15	23	20	29	15.4	891
INORGANIC COMPOUNDS						

TABLE 8

CHEMICALS DETECTED IN OFF-SITE SOIL BORINGS

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
Aluminum	3	3	3.6	3.6	6530	12500
Antimony	1	3	4.5	4.6	ND	13.1
Arsenic	3	3	0.48	0.48	8.8	18.7
Barium	3	3	0.24	0.24	155	280
Beryllium	3	3	0.24	0.24	0.64	0.91
Cadmium	2	3	0.71	0.73	2	3.3
Calcium	3	3	3.3	3.4	4560	18900
Chromium	3	3	0.71	0.73	22.3	128
Cobalt	3	3	0.71	0.73	9.6	13.3
Copper	3	3	0.71	0.73	73.4	192
Iron	3	3	0.71	0.73	16000	38700
Lead	3	3	0.48	0.48	29.7	213
Magnesium	3	3	5.5	5.6	3130	6020
Manganese	3	3	0.24	0.24	344	775
Mercury	3	3	0.12	0.12	0.4	1.8
Nickel	3	3	5.7	5.8	35.7	60.8
Potassium	3	3	100	100	736	1780
Selenium	1	3	0.48	0.48	ND	0.52
Silver	3	3	0.48	0.48	1.6	10.2
Sodium	3	3	4.3	4.4	184	294
Vanadium	3	3	0.71	0.73	10	24.5
Zinc	3	3	1.4	1.5	331	1120
Cyanide	2	3	0.3	0.3	0.73	3.7

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 9

CHEMICALS DETECTED IN CRANE-DEMING SOIL

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Tetrachloroethene	2	7	11	12	1	2
Toluene	3	7	11	12	2	3
SEMI-VOLATILE COMPOUNDS						
Naphthalene	1	6	380	390	ND	28
2-Methylnaphthalene	2	6	380	390	29	59
Phenanthrene	3	6	380	390	35	100
Fluoranthene	3	6	380	390	29	47
Pyrene	3	6	380	390	25	66
Butylbenzylphthalate	1	6	380	390	ND	32
Benzo(b)fluoranthene	1	6	380	390	ND	36
Benzo(k)fluoranthene	1	6	380	390	ND	30
Benzo(a)pyrene	1	6	380	390	ND	34
OTHER COMPOUNDS						
Photomirex	2	7	22	24	0.599	5.65
Mirex	4	7	20	22	6.39	100

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 10

CHEMICALS DETECTED IN RAILROAD TRACK TEST PIT SOIL

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Methylene chloride	2	11	5.5	150	71	74
1,2-Dichloroethene (total)	1	11	5.5	150	ND	12
Chloroform	1	11	5.5	150	ND	2
1,2-Dicholoethane	2	11	5.5	150	9	250
1,1,2,2-Tetrachloroethane	2	11	5.5	150	2	710
Trichloroethene	5	11	5.5	150	2	110
1,1,2-Trichloroethane	1	11	5.5	150	ND	110
Benzene	1	11	5.5	150	ND	510
Tetrachloroethene	3	11	5.5	150	4	98
Toluene	1	11	5.5	150	ND	60
Chlorobenzene	1	11	5.5	150	ND	25
SEMI-VOLATILE COMPOUNDS						
1,4-Dichlorobenzene	3	10	360	800	120	700
1,2-Dichlorobenzene	4	10	360	800	130	34000
2,4-Dichlorophenol	1	9	360	800	ND	190
1,2,4-Trichlorobenzene	1	9	360	800	ND	290
Naphthalene	8	11	360	800	73	850
2-Methylnaphthalene	8	11	360	800	110	880
Dibenzofuran	5	10	360	800	110	500
Diethylphthalate	1	9	360	800	ND	57
N-Nitrosodiphenylamine	3	10	360	800	59	750
Phenanthrene	8	11	360	800	69	1100
Anthracene	2	10	360	800	43	51
Fluoranthene	7	11	360	800	58	420
Pyrene	6	11	360	800	53	360
Benzo(a)anthracene	6	11	360	800	40	180
Chrysene	7	11	360	800	59	330
Bis(2-ethylhexyl)phthalate	2	9	360	800	49	160
Benzo(b)fluoranthene	7	11	360	800	110	540
Benzo(k)fluoranthene	7	11	360	800	110	540
Benzo(a)pyrene	5	11	360	800	47	180
Indeno(1,2,3-cd)pyrene	4	11	360	800	100	200
Benzo(g,h,i)perylene	4	11	360	800	93	160
Diphenylsulfone	6	9	360	800	130	90000
OTHER COMPOUNDS						
Kepone	1	11	78	110	ND	65.3
Photomirex	3	11	23	32	3.15	44.8
Mirex	9	11	21	1100	11.3	2230

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

TABLE 10

CHEMICALS DETECTED IN RAILROAD TRACK TEST PIT SOIL

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 11

CHEMICALS DETECTED IN MIDDLE FORK LITTLE BEAVER CREEK SURFACE WATER

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/l)		Range of Detected Concentrations (ug/l)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Chloromethane	1	8	1.5	10	ND	3
SEMI-VOLATILE COMPOUNDS						
Bis(2-ethylhexyl)phthalate	6	22	2	10	0.6	6
Diphenylsulfone	1	22	2	10	ND	2

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 12

CHEMICALS DETECTED IN SEDIMENT FROM MIDDLE FORK LITTLE BEAVER CREEK

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Acetone	4	6	12	13	27	80
1,2-Dichloroethane	1	6	5.5	6.5	ND	2
2-Butanone	1	6	11	13	ND	10
1,2-Dichloropropane	1	6	5.5	6.5	ND	18
SEMI-VOLATILE COMPOUNDS						
Phenol	2	24	350	550	120	160
4-Methylphenol	6	24	350	550	230	2800
Benzoic acid	2	24	1750	2800	210	430
Naphthalene	3	24	350	550	51	140
2-Methylnaphthalene	4	24	350	550	52	100
Acenaphthene	1	24	350	550	ND	100
Dibenzofuran	1	24	350	550	ND	180
Fluorene	1	24	350	550	ND	230
Phenanthrene	17	24	350	550	50	1800
Anthracene	1	24	350	550	ND	340
Di-n-butylphthalate	3	24	350	550	43	74
Fluoranthene	18	24	350	550	46	1100
Pyrene	15	24	350	550	46	790
Benzo(a)anthracene	10	23	350	550	41	480
Chrysene	14	24	350	550	45	530
Bis(2-ethylhexyl)phthalate	8	24	130	770	72	1800
Benzo(b)fluoranthene	17	24	350	550	52	680
Benzo(k)fluoranthene	17	24	350	550	52	680
Benzo(a)pyrene	13	24	350	550	38	310
Indeno(1,2,3-cd)pyrene	4	24	350	550	47	150
Benzo(g,h,i)perylene	5	24	350	550	41	120
Diphenylsulfone	2	49	350	910	55	170
PESTICIDES						
Heptachlor	1	27	2.6	215	ND	9.4
4,4'-DDE	1	26	5.3	430	ND	280
4,4'-DDT	1	27	5.3	430	ND	250
OTHER COMPOUNDS						
Photomirex	7	52	22.9	5590	0.479	7.38
Mirex	34	52	20.7	5070	4.26	2820

Background samples (#1, #29, #30, #47, and #50) were not included for purposes of determining frequency of detection.

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

TABLE 12

CHEMICALS DETECTED IN SEDIMENT FROM MIDDLE FORK LITTLE BEAVER CREEK

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 13

CHEMICALS DETECTED IN FISH FROM MIDDLE FORK LITTLE BEAVER CREEK

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
VOLATILE COMPOUNDS						
Methylene chloride	2	7	5	69	19	310
Acetone	5	7	10	210	120	820
2-Butanone	3	7	10	15	9	57
Benzene	1	7	5	5	ND	2
Tetrachloroethene	1	7	5	5	ND	7
Toluene	2	7	5	24	11	16
Ethylbenzene	3	7	5	5	4	5
Xylene (total)	1	7	5	5	ND	20
SEMI-VOLATILE COMPOUNDS						
Phenol	2	10	59	20000	93	380
Benzoic acid	2	18	59	20000	58	3300
4-Chloro-3-methylphenol	1	16	59	20000	ND	1400
Dimethylphthalate	1	49	59	20000	ND	210
N-Nitrosodiphenylamine	15	49	59	20000	54	1000
Di-n-butylphthalate	3	47	59	20000	50	1400
Butylbenzylphthalate	1	47	59	20000	ND	360
PESTICIDES						
Beta-BHC	1	46	7.3	80	ND	8.8
Gamma-BHC (Lindane)	1	45	7.3	80	ND	54
Aldrin	1	45	7.3	80	ND	8.2
Endosulfan I	2	42	7.3	80	30	67
4,4'-DDE	10	46	14.6	160	2.7	39
Endrin	2	45	14.6	160	13	49
alpha-Chlordane	3	46	7.3	80	5.9	14
OTHER COMPOUNDS						
Photomirex	37	55	9.54	162	0.35	390
Mirex	54	56	17.14	4490	5.2	6150

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

"ND" indicates that the chemical was detected in only one sample. Therefore, this detected value was placed in the maximum detected column and a ND or nondetect was placed in the minimum detected column.

TABLE 14

CHEMICALS DETECTED IN MIDDLE FORK LITTLE BEAVER CREEK FLOODPLAIN SOIL

CHEMICAL	Detection Frequency		Range of Reported Quantitation Limits (ug/kg)		Range of Detected Concentrations (ug/kg)	
	Number of Detects	Number of Samples	Minimum	Maximum	Minimum	Maximum
OTHER COMPOUNDS						
Photomirex	11	28	22.6	3960	2.5	132
Mirex	18	28	21.4	3400	16.4	4540

Minimum detected concentration could be less than minimum quantitation limit because of "J" or estimated values.

TABLE 15

REDUCTION IN NUMBER OF CHEMICALS TO BE QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	ENVIRON FREQUENCY OF DETECTION	ENVIRON ESSENTIAL NUTRIENTS	TOXICITY VALUES UNAVAILABLE	ENVIRON CONCENTRATION/ TOXICITY SCREEN
	VOLATILE COMPOUNDS				
1	1,1-Dichloroethene				x
2	1,1,1-Trichloroethane				x
3	1,1,2-Trichloroethane				
4	1,1,2,2-Tetrachloroethane				
5	1,2-Dichloroethane				
6	1,2-Dichloroethene (total)				
7	1,2-Dichloropropane				x
8	2-Butanone				x
9	Acetone				
10	Benzene				
11	Bromoform				x
12	Carbon Disulfide	x			
13	Carbon Tetrachloride				
14	Chlorobenzene				
15	Chloroethane	x			
16	Chloroform				x
17	Chloromethane				
18	Dibromochloromethane				x
19	Ethylbenzene				x
20	Methylene Chloride	x			
21	Styrene				x
22	Tetrachloroethene				
23	Toluene				x
24	Trichloroethene				
25	Vinyl Chloride				

TABLE 15

REDUCTION IN NUMBER OF CHEMICALS TO BE QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	ENVIRON FREQUENCY OF DETECTION	ENVIRON ESSENTIAL NUTRIENTS	TOXICITY VALUES UNAVAILABLE	ENVIRON CONCENTRATION/ TOXICITY SCREEN
26	Xylene (total)				x
	SEMIVOLATILE COMPOUNDS				
27	1,2-Dichlorobenzene				
28	1,2,4-Trichlorobenzene				x
29	1,3-Dichlorobenzene			x	
30	1,4-Dichlorobenzene				x
31	2-Chlorophenol				x
32	2-Methylnaphthalene			x	
33	2-Methylphenol	x			
34	2-Nitroaniline				x
35	2-Nitrophenol			x	
36	2,4-Dichlorophenol				
37	2,4-Dimethylphenol	x			
38	2,4,6-Trichlorophenol				x
39	2,6-Dinitrotoluene	x			
40	3-Nitroaniline	x			
41	4-Chloro-3-Methylphenol	x			
42	4-Methylphenol				
43	Acenaphthene				x
44	Acenaphthylene			x	
45	Anthracene				x
46	Benzoic Acid				x
47	Benzo(a)Anthracene				
48	Benzo(a)Pyrene				
49	Benzo(b)Fluoranthene				

TABLE 15

REDUCTION IN NUMBER OF CHEMICALS TO BE QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	ENVIRON FREQUENCY OF DETECTION	ENVIRON ESSENTIAL NUTRIENTS	TOXICITY VALUES UNAVAILABLE	ENVIRON CONCENTRATION/ TOXICITY SCREEN
50	Benzo(g,h,i)Perylene	x			
51	Benzo(k)Fluoranthene				
52	Benzyl Alcohol				x
53	bis(2-Chloroethoxy)Methane	x			
54	bis(2-Chloroethyl)Ether	x			
55	bis(2-Ethylhexyl)Phthalate				
56	Butylbenzylphthalate	x			
57	Carbazole	x			
58	Chrysene				x
59	Dibenzofuran			x	
60	Diethylphthalate				x
61	Dimethylphthalate	x			
62	Di-n-Butylphthalate	x			
63	Di-n-Octylphthalate	x			
64	Diphenylsulfone			x	
65	Fluoranthene				x
66	Fluorene				x
67	Hexachlorobenzene				
68	Hexachlorobutadiene				
69	Hexachlorocyclopentadiene	x			
70	Hexachloroethane				
71	Indeno(1,2,3-cd)Pyrene				
72	Naphthalene				x
73	Nitrobenzene	x			
74	N-Nitrosodiphenylamine				
75	N-Nitroso-di-n-Propylamine	x			

TABLE 15

REDUCTION IN NUMBER OF CHEMICALS TO BE QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	ENVIRON FREQUENCY OF DETECTION	ENVIRON ESSENTIAL NUTRIENTS	TOXICITY VALUES UNAVAILABLE	ENVIRON CONCENTRATION/ TOXICITY SCREEN
76	Pentachlorophenol	x			
77	Phenanthrene			x	
78	Phenol				x
79	Pyrene				
	PESTICIDES				
80	4,4'-DDD				x
81	4,4'-DDE	x			
82	4,4'-DDT				
83	Aldrin	x			
84	alpha-BHC	x			
85	alpha-Chlordane	x			
86	beta-BHC	x			
87	delta-BHC	x			
88	Dieldrin				
89	Endosulfan I	x			
90	Endosulfan II	x			
91	Endosulfan sulfate	x			
92	Endrin				x
93	Endrin aldehyde	x			
94	gamma-BHC (Lindane)	x			
95	gamma-Chlordane				x
96	Heptachlor				x
97	Methoxychlor				x

TABLE 15

REDUCTION IN NUMBER OF CHEMICALS TO BE QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	ENVIRON FREQUENCY OF DETECTION	ENVIRON ESSENTIAL NUTRIENTS	TOXICITY VALUES UNAVAILABLE	ENVIRON CONCENTRATION/ TOXICITY SCREEN
	OTHER				
98	Kepone				x
99	Mirex				
100	Photomirex				
	DIOXINS AND FURANS				
101	1,2,3,4,6,7,8-HpCDD				x
102	1,2,3,4,6,7,8-HpCDF				x
103	1,2,3,4,7,8-HxCDF				x
104	1,2,3,4,7,8,9-HpCDF				x
105	1,2,3,6,7,8-HxDCF				x
106	1,2,3,7,8-PeCDF				x
107	2,3,4,6,7,8-HxCDF				x
108	2,3,4,7,8-PeCDF				x
109	2,3,7,8-TCDD				x
110	2,3,7,8-TCDF				x
111	OCDD				x
112	OCDF				x
113	Total HpCDDs			x	
114	Total HpCDFs			x	
115	Total HxCDDs			x	
116	Total HxCDFs			x	
117	Total PeCDDS			x	
118	Total PeCDFs			x	
119	Total TCDD			x	
120	Total TCDFs			x	

TABLE 15

REDUCTION IN NUMBER OF CHEMICALS TO BE QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	ENVIRON FREQUENCY OF DETECTION	ENVIRON ESSENTIAL NUTRIENTS	TOXICITY VALUES UNAVAILABLE	ENVIRON CONCENTRATION/ TOXICITY SCREEN
	INORGANICS				
121	Aluminum			x	
122	Antimony				x
123	Arsenic				
124	Barium				x
125	Beryllium				
126	Cadmium				
127	Calcium		x		
128	Chromium				x
129	Cobalt			x	
130	Copper		x		
131	Iron		x		
132	Lead			x	
133	Magnesium		x		
134	Manganese		x		
135	Mercury				x
136	Nickel				x
137	Potassium		x		
138	Selenium				x
139	Silver				x
140	Sodium		x		
141	Thallium			x	
142	Vanadium				x
143	Zinc		x		
144	Cyanide				x

TABLE 15

REDUCTION IN NUMBER OF CHEMICALS TO BE QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

CHEMICAL	ENVIRON FREQUENCY OF DETECTION	ENVIRON ESSENTIAL NUTRIENTS	TOXICITY VALUES UNAVAILABLE	ENVIRON CONCENTRATION/ TOXICITY SCREEN
TOTALS	31	8	19	53

NOTE: "x" indicates chemical eliminated from quantitative risk assessment because of listed criterion.

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TABLE 16

CHEMICALS QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL
	VOLATILE COMPOUNDS										
1	1,1,2-Trichloroethane				x						
2	1,1,2,2-Tetrachloroethane	x		x	x	x					
3	1,2-Dichloroethane	x									
4	1,2-Dichloroethene (total)	x			x						
5	Acetone				x						
6	Benzene	x		x	x						
7	Carbon Tetrachloride				x		x				
8	Chlorobenzene	x									
9	Chloromethane							x			
10	Tetrachloroethene	x		x	x						
11	Trichloroethene	x		x	x						
12	Vinyl Chloride	x									
	SEMIVOLATILE COMPOUNDS										
13	1,2-Dichlorobenzene	x			x	x					
14	2,4-Dichlorophenol	x			x						
15	4-Methylphenol									x	
16	Benzo(a)Anthracene					x				x	
17	Benzo(a)Pyrene		x			x				x	
18	Benzo(b)Fluoranthene					x				x	
19	Benzo(k)Fluoranthene					x				x	
20	bis(2-Ethylhexyl)Phthalate					x	x	x			
21	Hexachlorobenzene		x	x							
22	Hexachlorobutadiene	x		x							
23	Hexachloroethane	x		x	x						
24	Indeno(1,2,3-cd)Pyrene					x				x	

TABLE 16

CHEMICALS QUANTITATIVELY CONSIDERED IN RISK ASSESSMENT

	CHEMICAL	GROUND WATER	ON-SITE SEDIMENT	ON-SITE SOIL	ON-SITE SURFACE WATER	OFF-SITE SOIL	AIR	MFLBC SURFACE WATER	MFLBC FISH	MFLBC SEDIMENT	MFLBC FLOOD PLAIN SOIL
25	N-Nitrosodiphenylamine						x				
26	Pyrene					x					
	PESTICIDES										
27	4,4'-DDT					x				x	
28	Dieldrin			x		x					
	OTHER										
29	Mirex	x	x	x	x	x	x		x	x	x
30	Photomirex								x		x
	INORGANICS										
31	Arsenic	x									
32	Beryllium	x									
33	Cadmium	x									
	TOTALS	16	3	9	12	12	4	2	2	8	2

NOTE: "x" indicates that chemical was quantitatively considered in the risk assessment for the environmental medium; no entry indicates chemical was not quantitatively considered in the risk assessment for the environmental medium.

TABLE 17
Potential Exposure Pathways Quantitatively Assessed at the Ruetgers-Nease Salem Site

Exposure Medium/ Exposure Route	Potentially Exposed Population						
	On-site			Areas Adjacent to Site		Locations Along the MFLBC	
	Trespasser	Worker	Resident	Worker	Resident	Recreational Visitor	Resident
Ingestion of Ground Water	-	F	F	-	-	-	-
Ingestion of Soil	C,F	F	F	C,F	C,F	-	C,F
Inhalation of Air	C,F	F	F	-	-	-	-
Ingestion of Surface Water	C,F	-	-	-	-	C,F	-
Ingestion of Sediments	C,F	-	-	-	-	C,F	-
Ingestion of Fish	-	-	-	-	-	C,F	-
Ingestion of Game	-	-	-	-	-	C,F	-
Ingestion of Vegetables	-	-	F	-	C,F	-	C,F
Ingestion of Beef and Milk	-	-	-	-	-	-	F

C,F Indicates that potential exposure is possible under both current and hypothetical future exposure scenarios.
F Indicates that potential exposure is possible only under the hypothetical future exposure scenario.
-- Indicates not a complete exposure pathway for this receptor population.

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TABLE 18
Reasonable Maximum Exposure Concentrations
for Chemicals in Ground Water

CHEMICAL	ROUND 1 (ug/l) D12	T2	RW1	ROUND 2 (ug/l) D12	T2	RW1	CONCENTRATION (ug/l)
VOLATILE COMPOUNDS							
1,1,2,2-Tetrachloroethane	13,000	5,300	9,300	11,000	5,300	10,000	13,000
1,2-Dichloroethane	<250	1,000	<10,000	<2,500	<10,000	<5,000	1,000
1,2-Dichloroethene (total)	1,200	15,400	<10,000	1,700	<10,000	2,400	15,400
Benzene	1,900	4,100	4,100	1,300	5,100	4,400	5,100
Chlorobenzene	480	270	<10,000	570	<10,000	<5,000	570
Tetrachloroethene	27,000	66,000	77,000	38,000	100,000	92,000	100,000
Trichloroethene	11,000	17,000	8,400	8,500	19,000	8,100	19,000
SEMIVOLATILE COMPOUNDS							
1,2-Dichlorobenzene	31,000	12,000	14,000	28,000	8,000	16,000	31,000
2,4-Dichlorophenol	18	35	11	14	47	15	47
Hexachlorobutadiene	110	44	26	36	42	32	110
Hexachloroethane	470	420	420	190	300	410	470
OTHER							
Mirex	239.6	16.4	24.7	24.9	14.9	180	239.6
INORGANICS							
Arsenic	58	<160	NA	NA	5.6	NA	58
Beryllium	78	<3	NA	NA	1.02	NA	78
Cadmium	123	<8.5	NA	NA	4.33	NA	123

NA = Chemical not analyzed
<250 = Chemical not detected at the given detection limit

Although vinyl chloride was selected as a chemical for evaluation in the ground water medium, it was not detected in wells D12, T2, or RW1. This chemical was therefore not included in these calculations.

TABLE 19
Reasonable Maximum Exposure Concentrations
for Chemicals in On-Site Soil Outside Fenceline

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	11	13.8	26.6	110.0	26.6
Benzene (1)	11	3.9	5.2	2.0	2.0
Tetrachloroethene	11	28.5	103.2	260.0	103.2
Trichloroethene	11	6.8	11.8	7.0	7.0
SEMIVOLATILE COMPOUNDS					
Hexachlorobenzene	11	651.7	1509.1	1500.0	1500.0
Hexachlorobutadiene (2)	11	452.1	767.3	290.0	290.0
Hexachloroethane (2)	11	442.1	763.1	180.0	180.0
OTHER					
Mirex	11	15324.1	31999640.5	92800.0	92800.0

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for on-site soil samples outside the fenceline include the following:
TP-24-0.5, TP-25-0.5, TP-26-0.5, TP-27-0.5, TP-28-0.5, TP-29-0.5, SB-26-3, SB-27-3, SB-28-1, SB-29-3, SB-30-3

Although Dieldrin was selected as a chemical for evaluation in the surface soil medium, it was not detected in any on-site surficial soil samples outside the fence. This chemical was therefore not included with these calculations.

(1) Benzene was detected in one sample at a concentration of 2.0 ug/kg. The reported mean value reflects an average of this detected concentration and one-half of detection limits for non-detected samples.

(2) The maximum detected concentrations of hexachlorobutadiene and hexachloroethane are lower than one half the detection limit for some non-detected samples. Therefore, the maximum detected concentration is lower than the reported mean concentration.

TABLE 20
Reasonable Maximum Exposure Concentrations
for Chemicals in On-Site Soil Borings and Test Pits

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	42	3396.3	8224.9	120000.0	8224.9
Benzene	42	1078.0	2428.9	33000.0	2428.9
Tetrachloroethene	42	8514.8	19520.7	270000.0	19520.7
Trichloroethene	42	1036.6	2385.7	33000.0	2385.7
SEMIVOLATILE COMPOUNDS					
Hexachlorobenzene	41	1609.6	2959.3	4900.0	2959.3
Hexachlorobutadiene (1)	37	345.1	1372.5	810.0	810.0
Hexachloroethane	40	1674.4	3858.6	2400.0	2400.0
PESTICIDES					
Dieldrin	41	308.5	1012.7	3000.0	1012.7
OTHER					
Mirex	42	73866.4	688273.8	2080000.0	688273.8

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for on-site soil samples include the following:
TP-01-0.5, TP-02-0.5, TP-04-0.5, TP-05-0.5, TP-06-0.5, TP-07-0.5, TP-08-0.5, TP-09-0.5, TP-10-0.5, TP-11-0.5, TP-12-0.5, TP-13-0.5, TP-14-0.5, TP-15-0.5, TP-16-0.5, TP-17-0.5, TP-18-0.5, TP-19-0.5, TP-20-0.5, TP-21-0.5, TP-22-0.5, TP-23-0.5, TP-24-0.5, TP-25-0.5, TP-26-0.5, TP-27-0.5, TP-28-0.5, TP-29-0.5, SB-17-4, SB-18-3. SB-19-3, SB-20R-3, SB-21-3, SB-22-3, SB-23-3, SB-24-3, SB-25-3, SB-26-3, SB-27-3, SB-28-1, SB-29-3, SB-30-3

(1) Hexachlorobutadiene was not detected in samples SB-17-4, SB-19-3, and SB-21-3. Because of unusually high detection limits, one-half the detection limit was not used as a proxy concentration for these samples. These samples were instead omitted from the calculations for hexachlorobutadiene (USEPA 1988, p. 5-10).

TABLE 21
Reasonable Maximum Exposure Concentrations
for Chemicals in Crane-Deming Soils

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
SEMIVOLATILE COMPOUNDS					
Benzo(a)Pyrene (1)	1	34.0	NA	34.0	34.0
Benzo(b)Fluoranthene (1)	1	36.0	NA	36.0	36.0
Benzo(k)Fluoranthene (1)	1	30.0	NA	30.0	30.0
Pyrene	3	44.3	159.7	66.0	66.0
OTHER					
Mirex	3	40.9	99023.8	100.0	100.0

NA Not Applicable -- calculation of a statistical distribution and confidence limit on the basis of a single sample is not possible.

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for Crane-Deming soil samples include the following: TP-31-0.5, TP-30-0.5, and SB-35-0.5.

Although 1,1,2,2-Tetrachloroethane, 1,2-dichlorobenzene, benzo(a)anthracene, bis(2-ethylhexyl)phthalate, indeno(1,2,3-cd)pyrene, 4,4'-DDT, and dieldrin were selected as chemicals for evaluation in the Crane-Deming soil medium, they were not detected in any samples. These chemicals were therefore not included with the above calculations.

(1) Benzo(a)pyrene, benzo(b)fluoranthene, and benzo(k)fluoranthene were not detected in samples TP-31-0.5 and SB-35-0.5. Because of unusally high detection limits, one half the detection limit was not used as a proxy concentration for these samples. These samples were instead omitted from the calculation (USEPA 1989, p. 5-10).

TABLE 22
Reasonable Maximum Exposure Concentration
for Chemicals in Off-Site Soil Borings

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
SEMIVOLATILE COMPOUNDS					
Benzo(a)anthracene (1)	9	185.3	253.7	64.0	64.0
Benzo(a)pyrene (1)	9	184.1	272.3	53.0	53.0
Benzo(b)fluoranthene (1)	9	160.0	270.4	98.0	98.0
Benzo(k)fluoranthene (1)	9	160.0	270.4	98.0	98.0
bis(2-Ethylhexyl)phthalate	10	145.4	497.9	560.0	497.9
Indeno(1,2,3-cd)pyrene (1)	9	183.4	282.2	47.0	47.0
Pyrene (1)	9	155.6	311.2	88.0	88.0
PESTICIDES					
4,4'-DDT	10	20.0	33.2	110.0	33.2
Dieldrin (1)	10	9.6	10.8	6.2	6.2
OTHER					
Mirex	10	266.8	639.3	832.0	639.3

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for off-site soil samples include the following:
SB-01-0.5, SB-02-0.5, SB-03-0.5, SB-04-0.5, SB-05-0.5, SB-07-0.5, SB-08-0.5, SB-09-0.5, SB-11-0.5, SB-12-0.5

Although 1,1,2,2-Tetrachloroethane was selected as a chemical for evaluation in the off-site soil boring medium, it not analyzed in any surficial soil samples. This chemical was therefore not included with these calculations.

Although 1,2-Dichlorobenzene was selected as a chemical for evaluation in the off-site soil boring medium, it was not detected in any surficial soil samples. This chemical was therefore not included with these calculations.

(1) The maximum detected concentrations for these chemicals are lower than one half the detection limit for some of the non-detected samples. Therefore the maximum detected concentrations for these chemicals are less than the reported mean concentrations.

TABLE 23
Reasonable Maximum Exposure Concentrations
for Chemicals in Floodplain Soil

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
OTHER					
Photomirex	28	20.3	27.2	132.0	27.2
Mirex	28	565.0	4108.8	4540.0	4108.8

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for floodplain soil samples include the following:
SS-10-01, SS-10-02, SS-10-03, SS-10-04, SS-12-01, SS-12-02, SS-12-03, SS-12-14, SS-17-01, SS-17-02, SS-17-03, SS-17-04, SS-19A-01, SS-19A-02, SS-19A-03, SS-19A-04, SS-19B-01, SS-19B-02, SS-19B-03, SS-19B-04, SS-27-01, SS-27-02, SS-27-03, SS-27-04, SS-43-01, SS-43-02, SS-43-03, SS-43-04

TABLE 24
Reasonable Maximum Exposure Concentrations
for Chemicals in Air

CHEMICAL	Number of Samples	Mean Concentration (ng/m^3)	Upper 95% Confidence Limit on Mean (ng/m^3)	Maximum Detected Concentration (ng/m^3)	RME Concentration (ng/m^3)
VOLATILE COMPOUNDS					
Carbon Tetrachloride	8	NC	NC	16.7	16.7
SEMIVOLATILE COMPOUNDS					
bis(2-Ethylhexyl)Phthalate	6	NC	NC	25500.0	25500.0
N-Nitrosodiphenylamine	6	NC	NC	2200.0	2200.0
OTHER					
Mirex	6	NC	NC	47.0	47.0

NC Not Calculated -- The mean concentration and the upper 95% confidence limit on the mean were not calculated because the maximum detected concentrations were used as the RME concentrations

Samples used for calculating the maximum detect and the RME values for air samples include the following:
AO-01-1004, AO-02-3406, AO-02-3442, AO-03-1-026, AO-04-3412, AO-05-3411, AO-06-3419, AO-06-3443, AO-01-024, AO-02-3516, AO-03-004, AO-04-2-3503, AO-05-3508, AO-06-3515, AO-06-3543, AO-01-3-022, AO-02-3-037, AO-03-3-003, AO-04-3-042, AO-05-034, AO-06-3-014, AO/AP-01-4-005, AO/AP-02-4-008, AO/AP-034-4-002, AO/AP-04-4-006, AO/AP-05-4-00, AO/AP-06-4-004

TABLE 25
Reasonable Maximum Exposure Concentrations
for Chemicals in On-Site Surface Water

	Number of Samples	Mean Concentration (ug/l)	Upper 95% Confidence Limit on Mean (ug/l)	Maximum Detected Concentration (ug/l)	RME Concentration (ug/l)
VOLATILE COMPOUNDS					
1,1,2-Trichloroethane	7	3.1	3.9	5.0	3.9
1,1,2,2-Tetrachloroethane	7	97.4	433697.6	470	470.0
1,2-Dichloroethene (total)	7	6.8	23.2	14	14.0
Acetone	7	34.1	432.1	170	170.0
Benzene	7	27.1	1110.4	140	140.0
Carbon Tetrachloride (1)	7	2.4	2.6	2	2.0
Tetrachloroethene	7	59.1	8657.0	340	340.0
Trichloroethene	7	27.3	1219.6	94	94.0
SEMIVOLATILE COMPOUNDS					
1,2-Dichlorobenzene	8	30.6	471.7	180	180.0
2,4-Dichlorophenol (2)	8	4.4	7.8	4	4.0
Hexachloroethane	8	4.5	8.1	5	5.0
OTHER					
Mirex	8	0.1	3.4	0.362	0.4

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for on-site surface water samples include the following:
SW-02, SW-03, SW-04, SW-55, SW-57, SW-58, SW-59, SW-01

(1) Carbon tetrachloride was detected in one sample at a concentration of 2.0 ug/l. The reported mean value reflects an average of this detected concentration and one-half of detection limits for non-detected samples.

(2) 2,4-Dichlorophenol was detected in one sample at a concentration of 4.0 ug/l. The reported mean value reflects an average of this detected concentration and one-half of detection limits for non-detected samples.

TABLE 26
Reasonable Maximum Exposure Concentrations
for Chemicals in Middle Fork Little Beaver Creek Surface Water

CHEMICAL	Number of Samples	Mean Concentration (ug/l)	Upper 95% Confidence Limit on Mean (ug/l)	Maximum Detected Concentration (ug/l)	RME Concentration (ug/l)
VOLATILE COMPOUNDS					
Chloromethane (1)	8	4.2	9.0	3.0	3.0
SEMIVOLATILE COMPOUNDS					
bis(2-Ethylhexyl)Phthalate	22	4.4	5.8	6.0	5.8

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for Middle Fork Little Beaver Creek surface water samples include the following:
SW-01, SW-02, SW-03, SW-04, SW-05, SW-6C, SW-07, SW-08, SW-13, SW-18, SW-20, SW-23, SW-28, SW-29, SW-30, SW-35, SW-40, SW-42, SW-47, SW-48, SW-50, SW-52

(1) Chloromethane was detected in one sample at a concentration of 3.0 ug/l. The reported mean value reflects an average of this detected concentration and one-half of detection limits for non-detected samples.

TABLE 27
Reasonable Maximum Exposure Concentrations
for Chemicals in On-Site Sediment

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
SEMIVOLATILE COMPOUNDS					
Benzo(a)Pyrene	7	296.7	1194.3	310.0	310.0
Hexachlorobenzene	7	995.1	17523.3	3000.0	3000.0
OTHER					
Mirex (1)	7	26954.7	NA	129000.0	129000.0

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for on-site sediment samples include the following:
SD-58, SD-55, SD-57, SD-56, SD-59, SD-53, SD-54

(1) NA - Not applicable: The upper 95th upper confidence limit on the mean for mirex, as calculated according to the Land equation for a lognormal distribution, does not represent a realistic physical quantity. This value was therefore not included.

TABLE 28

Reasonable Maximum Exposure Concentrations

for Chemicals in Sediment from Middle Fork Little Beaver Creek (Upstream of Lisbon Dam)

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
SEMIVOLATILE COMPOUNDS					
4-Methylphenol	16	499.2	557.7	2800	557.7
Benzo(a)Anthracene (1)	16	144.4	151.6	100	100.0
Benzo(a)Pyrene (1)	16	124.0	127.5	85	85.0
Benzo(b)Fluoranthene	16	137.6	173.0	260	173.0
Benzo(k)Fluoranthene	16	137.6	173.0	260	173.0
Indeno(1,2,3-cd)Pyrene (1)	16	163.6	169.0	71	71.0
PESTICIDES					
4,4'-DDT	16	75.2	191.0	250	191.0
OTHER					
Mirex	36	266.1	497.0	2820	497.0

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for upstream Middle Fork Little Beaver Creek sediment samples include the following:
SD-02, SD-03, SD-04, SD-05, SD-6C, SD-07, SD-10, SD-11, SD-12, SD-13, SD-14, SD-15, SD-16, SD-17, SD-17-02, SD-18, SD-19, SD-19A, SD-19B, SD-20, SD-21, SD-22, SD-23, SD-24, SD-25, SD-26, SD-27, SD-28, SD-31, SD-32, SD-33, SD-34, SD-35, SD-37, SD-38, SD-39

(1) The maximum detected concentrations for these chemicals are lower than one half the detection limit for some of the non-detected samples. Therefore, the maximum detected concentrations for these chemicals are lower than the reported mean concentrations.

TABLE 29
Reasonable Maximum Exposure Concentrations
for Chemicals in Sediment from Middle Fork Little Beaver Creek (Downstream of Lisbon Dam)

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
SEMIVOLATILE COMPOUNDS					
4-Methylphenol	8	452.8	950.1	2100	950.1
Benzo(a)anthracene	7	209.4	400.0	480	400.0
Benzo(a)pyrene	8	164.0	340.8	310	310.0
Benzo(b)fluoranthene	8	236.5	389.2	680	389.2
Benzo(k)fluoranthene	8	236.5	389.2	680	389.2
Indeno(1,2,3-cd)pyrene (1)	8	178.0	289.3	150	150.0
OTHER					
Mirex	11	11.0	12.4	13.875	12.4

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for downstream Middle Fork Little Beaver Creek sediment samples include the following:
SD-40, SD-41, SD-42, SD-43, SD-44, SD-45, SD-46, SD-48, SD-49, SD-51, SD-52

Although 4,4'-DDT was selected as a chemical for evaluation in Middle Fork Little Beaver Creek sediment, it was not detected in any samples downstream of the Lisbon Dam. This chemical is therefore not included with the calculations.

(1) The maximum detected concentration of indeno(1,2,3-cd)pyrene is 150 ug/kg. The reported mean value reflects an average of concentrations of detected samples and one half of detection limits for non-detected samples.

TABLE 30
Reasonable Maximum Exposure Concentrations
for Chemicals in Fish (Above and Below Lisbon Dam)

UPSTREAM FROM LISBON DAM

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
OTHER					
Mirex	16	322.2	1042.2	1820.0	1042.2
Photomirex (1)	15	9.4	18.0	28.8	18.0

DOWNSTREAM FROM LISBON DAM

CHEMICAL	Number of Samples	Mean Concentration (ug/kg)	Upper 95% Confidence Limit on Mean (ug/kg)	Maximum Detected Concentration (ug/kg)	RME Concentration (ug/kg)
OTHER					
Mirex	12	23.7	39.4	67.0	39.4
Photomirex (2)	12	5.4	15.2	3.1	3.1

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for fish tissue samples in the section of Middle Fork Little Beaver Creek upstream from the Lisbon dam include the following:
FI-6C-UT, FI-07F-LT, FI-08-UT, FI-09-UT, FI-13-UT, FI-15-UT, FI-18-UT, FI-20-UT, FI-23-UT, FI-28-UT, FI-30-UT, FI-35-UT, FI-37-UT, FI-39-UT, FI-39F-LT, FI-39F-UT

Samples used for calculating the mean, the upper 95th percent confidence limit, the maximum detect, and the RME values for fish tissue samples in the section of Middle Fork Little Beaver Creek downstream from the Lisbon dam include the following:
FI-40-UT, FI-42-UT, FI-44-UT, FI-45-UT, FI-47-UT, FI-48-UT, FI-48F-LT, FI-48F-UT, FI-49-UT, FI-50-UT, FI-51-UT, FI-52-UT

(1) Photomirex was not detected in sample FI-08-UT. Because of an unusually high detection limit, one half the detection limit is not used as a proxy concentration for this sample. This sample was instead omitted from the calculations for photomirex (USEPA 1989, p. 5-10).

(2) The maximum detected concentration of photomirex is 3.1 ug/kg. The reported mean value reflects an average of concentrations of detected samples and one half of detection limits for non-detected samples.

TABLE 31
Chemical Concentrations
in Produce

CHEMICAL	RME Soil Concentration (ug/kg)	Concentration in Produce (mg/kg)
On-Site Soil		
VOLATILE COMPOUNDS		
1,1,2,2-Tetrachloroethane	8224.9	7.6E-03
Benzene	2428.9	2.3E-03
Tetrachloroethene	19520.7	1.8E-02
Trichloroethene	2385.7	2.2E-03
SEMIVOLATILE COMPOUNDS		
Hexachlorobenzene	2959.3	2.8E-03
Hexachlorobutadiene	810.0	7.5E-04
Hexachloroethane	2400.0	2.2E-03
PESTICIDES		
Dieldrin	1012.7	9.4E-04
OTHER		
Mirex	688273.8	6.4E-01
Off-Site Soil		
SEMIVOLATILE COMPOUNDS		
Benzo(a)Anthracene	64.0	6.0E-05
Benzo(a)Pyrene	53.0	4.9E-05
Benzo(b)Fluoranthene	98.0	9.1E-05
Benzo(k)Fluoranthene	98.0	9.1E-05
bis(2-Ethylhexyl)Phthalate	497.9	4.6E-04
Indeno(1,2,3-cd)Pyrene	47.0	4.4E-05
Pyrene	88.0	8.2E-05
PESTICIDES		
4,4'-DDT	33.2	3.1E-05
Dieldrin	6.2	5.8E-06
OTHER		
Mirex	639.3	5.9E-04
Floodplain Soil		
OTHER		
Photomirex	27.2	2.5E-05
Mirex	4108.8	3.8E-03

TABLE 32
Chemical Concentrations
in Beef

CHEMICAL	RME Soil Concentration (ug/kg)	Concentration in Beef (mg/kg)
Floodplain Soil		
OTHER		
Photomirex	27.2	0.003
Mirex	4108.8	0.44

TABLE 33
Chemical Concentrations
in Milk

CHEMICAL	RME Soil Concentration (ug/kg)	Concentration in Milk (mg/kg)
Floodplain Soil		
OTHER		
Photomirex	27.2	0.00003
Mirex	4108.8	0.005

TABLE 34
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
– On-site Trespasser –
Current and Future Use

Exposure Medium/ Exposure Route	Current Use		Future Use	
	Cancer Risk Estimate	Noncancer HI Estimate	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Soil	1×10^{-6}	0.07	7×10^{-6}	0.5
Inhalation of Air	3×10^{-7}	0.008	3×10^{-7}	0.008
Ingestion of Surface Water	2×10^{-7}	0.0008	2×10^{-7}	0.0008
Ingestion of Sediment	4×10^{-7}	0.003	4×10^{-7}	0.003
	Total Cancer Risk: 2×10^{-6}	Cumulative HI: 0.08	Total Cancer Risk: 8×10^{-6}	Cumulative HI: 0.5

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 35
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
-- Worker at Locations Adjacent to Site --
Current and Future Use

Exposure Medium/ Exposure Route	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Soil	6×10^{-8}	0.0003
	Total Cancer Risk: 6×10^{-8}	Cumulative HI: 0.0003

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 36
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
– Residents at Locations Adjacent to Site –
Current and Future Use

Exposure Medium/ Exposure Route	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Soil	2×10^{-6}	0.04
Ingestion of Vegetables	2×10^{-7}	0.002
	Total Cancer Risk: 2×10^{-6}	Cumulative HI: 0.04

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 37
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
– Recreational Visitor –
Current Use

Exposure Medium/ Exposure Route	Upstream of Advisory		Downstream of Advisory	
	Cancer Risk Estimate	Noncancer HI Estimate	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Surface Water	4×10^{-9}	0.00002	4×10^{-9}	0.00002
Ingestion of Sediments	3×10^{-7}	0.0065	2×10^{-6}	0.001
Ingestion of Fish	6×10^{-6}	0.1	3×10^{-6}	0.06
Ingestion of Game	5×10^{-8}	0.001	5×10^{-8}	0.001
	Total Cancer Risk: 6×10^{-6}	Cumulative HI: 0.1	Total Cancer Risk: 5×10^{-6}	Cumulative HI: 0.06

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 38
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
– Flood plain Resident –
Current and Future Use

Exposure Medium/ Exposure Route	Current Use		Future Use	
	Cancer Risk Estimate	Noncancer HI Estimate	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Soil	3×10^{-6}	0.3	3×10^{-6}	0.3
Ingestion of Vegetables	5×10^{-7}	0.01	5×10^{-7}	0.01
Ingestion of Beef	NA	NA	6×10^{-5}	1
Ingestion of Milk	NA	NA	4×10^{-6}	0.3
	Total Cancer Risk: 4×10^{-6}	Cumulative HI: 0.3	Total Cancer Risk: 7×10^{-5}	Cumulative HI: 2

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 39
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
– On-site Worker –
Future Use

Exposure Medium/ Exposure Route	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Ground Water	$> 1 \times 10^{-2}$	145
Ingestion of Soil	7×10^{-5}	2
Inhalation of Air	3×10^{-5}	0.3
	Total Cancer Risk: $> 1 \times 10^{-2}$	Cumulative HI: 147

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 40
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
– On-site Resident –
Future Use

Exposure Medium/ Exposure Route	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Ground Water	$> 1 \times 10^{-2}$	400
Ingestion of Soil	6×10^{-4}	44
Inhalation of Air	5×10^{-5}	0.4
Ingestion of Vegetables	9×10^{-5}	2
	Total Cancer Risk: $> 1 \times 10^{-2}$	Cumulative HI: 446

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 41
Estimated Cancer Risks and Noncancer Hazard Index Values[1]
– Recreational Visitor –
Future Use

Exposure Medium/ Exposure Route	Upstream of Advisory		Downstream of Advisory	
	Cancer Risk Estimate	Noncancer HI Estimate	Cancer Risk Estimate	Noncancer HI Estimate
Ingestion of Surface Water	7×10^{-9}	0.00004	7×10^{-9}	0.00004
Ingestion of Sediments	6×10^{-7}	0.01	2×10^{-6}	0.001
Ingestion of Fish	7×10^{-5}	2	3×10^{-6}	0.06
Ingestion of Game	5×10^{-8}	0.001	5×10^{-8}	0.001
	Total Cancer Risk: 7×10^{-5}	Cumulative HI: 2	Total Cancer Risk: 5×10^{-6}	Cumulative HI: 0.06

[1] Note: The cancer risk and hazard index (HI) values presented in this table are developed using risk assessment methods discussed in Chapter I (Section B) and Chapter VIII. The cancer risk values are upper bound risk estimates and do not represent actuarial risks. Similarly, noncancer risk assessment incorporates a number of conservative assumptions about exposure and toxicity; resulting HI values do not represent either probabalistic or actuarial risks.

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TABLE 42
MFLBC QHEI Scores and Use Attainability Status according to Rankin (1989)[a]

River Mile[b]	QHEI Score[c]	WWH Attainment Status[c]	EWH Attainment Status[c]
40.03	59	meets	does not meet
38.3	47	meets	does not meet
37.6-37.7	55.5	meets	does not meet
36.7	66	meets	meets
35.4	68	meets	meets
32.6-32.7	51.5	meets	does not meet
28.8	41	does not meet	does not meet
26.8-26.9	43	does not meet	does not meet
25.1	48	meets	does not meet
21.8	55.5	meets	does not meet
20.9	32	does not meet	does not meet
15.1	89	meets	meets
10.9	73	meets	meets
9.0	87	meets	meets
1.9	81	meets	meets

[a] A copy of the Ohio EPA MFLBC map (Appendix 1) locates the survey stations in the appropriate regions along MFLBC and includes the QHEI score and the use attainability. The figure also presents the location of the Salem WWTP, the Ruetgers-Nease Chemical Company, and Butterfield Creek for reference.

[b] River mile designation begins at the confluence with the West fork of Little Beaver Creek and moves upstream.

[c] See text for description of terminology.

TABLE 43
Selection of Chemicals for Evaluation in the Ecological Assessment[1]
(all concentrations are in ppm)

Chemical	Sediment[1a]		Water		Fish Tissue		Selected for Evaluation
	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	
Acetone	0.08	2.26[7]	ND[2]	NP[14]	0.82	NA[4]	NO[5,6]
1,2-Dichloroethane	0.002	0.035[3]	ND	NP	ND	NP	NO[5]
2-Butanone	0.01	0.639[7]	ND	NP	0.06	NA	NO[5,6]
1,2-Dichloropropane	0.018	1,448[15]	ND	NP	ND	NP	NO[5]
Phenol	0.160	0.99[15]	ND	NP	0.38	NA	NO[5,6]
4-Methylphenol	2.8	0.202	ND	NP	ND	NP	YES
Benzoic acid	0.43	0.624[16]	ND	NP	3.3	NA	NO[5,6]
Naphthalene	0.14	3.30[7]	ND	NP	ND	NP	NO[5]
2-Methylnaphthalene	0.10	0.12 - 94.2[8]	ND	NP	ND	NP	NO[8]
Acenaphthene	0.10	36.6[13]	ND	NP	ND	NP	NO[5,9]
Dibenzofuran	0.18	0.50[15]	ND	NP	ND	NP	NO[5,9]
Fluorene	0.23	28.3[15]	ND	NP	ND	NP	NO[5,9]
Phenanthrene	1.80	6.95[13]	ND	NP	ND	NP	NO[5]
Anthracene	0.34	32.2[15]	ND	NP	ND	NP	NO[5,9]

TABLE 43
Selection of Chemicals for Evaluation in the Ecological Assessment[1]
(all concentrations are in ppm)

Chemical	Sediment[1a]		Water		Fish Tissue		Selected for Evaluation
	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	
Di-n-butylphthalate	0.074	415[7]	ND	NP	1.4	NA	NO[6]
Fluoranthene	1.1	94.2[13]	ND	NP	ND	NP	NO[5]
Pyrene	0.79	65.6[13]	ND[2]	NP[14]	ND	NP	NO[5]
Benzo(a)anthracene	0.48	65.9[13]	ND	ND	ND	NP	NO[5]
Chrysene	0.53	52.7[15]	ND	NP	ND	NP	NO[5]
Bis(2-ethylhexyl)phthalate	1.8	5.99[3]	0.006	0.360[10]	ND	NP	NO[5]
Benzo(b)fluoranthene	0.68	65.3[15]	ND	NP	ND	NP	NO[5]
Benzo(k)fluoranthene	0.68	74.6[15]	ND	NP	ND	NP	NO[5]
Benzo(a)pyrene	0.31	53.2[13]	ND	NP	ND	NP	NO[5]
Indeno(1,2,3-cd)pyrene	0.15	80.6[15]	ND	NP	ND	NP	NO[5]
Benzo(g,h,i)perylene	0.12	79.9[15]	ND	NP	ND	NP	NO[5]
Diphenylsulfone	0.17	NA	0.002	NP	ND	NP	NO[6,9]
Heptachlor	0.009	0.008[7]	ND	NP	ND	NP	NO[9]
DDTr	0.53	0.061[7]	ND	NP	0.039	0.20	NO[9]

TABLE 43
Selection of Chemicals for Evaluation in the Ecological Assessment[1]
(all concentrations are in ppm)

Chemical	Sediment[1a]		Water		Fish Tissue		Selected for Evaluation
	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	
Photomirex	0.007	NA	ND	NP	0.390	NA	YES[11]
Mirex	2.82	2.4[15]	ND	NP	6.15	0.67	YES
Methylene chloride	ND	NP	ND	NP	0.31	NA	NO[6]
Benzene	ND	NP	ND	NP	0.002	NA	NO[6]
Tetrachloroethene	ND	NP	ND	NP	0.007	NA	NO[6]
Toluene	ND	NP	ND	NP	0.016	NA	NO[6]
Ethylbenzene	ND	NP	ND	NP	0.005	NA	NO[6]
Xylenes	ND	NP	ND	NP	0.02	NA	NO[6]
4-Chloro-3-methylphenol	ND	NP	ND	NP	1.4	NA	NO[6]
Dimethylphthalate	ND	NP	ND	NP	0.21	NA	NO[9]
N-nitrosodiphenylamine	ND	NP	ND	NP	1.0	NA	NO[6]
Butylbenzylphthalate	ND	NP	ND	NP	0.36	NA	NO[9]
Beta-BHC	ND	NP	ND	NP	0.009	NA	NO[9]
Lindane (Gamma-BHC)	ND	NP	ND	NP	0.054	NA	NO[9]

TABLE 43
Selection of Chemicals for Evaluation in the Ecological Assessment[1]
(all concentrations are in ppm)

Chemical	Sediment[1a]		Water		Fish Tissue		Selected for Evaluation
	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	
Aldrin	ND	NP	ND	NP	0.008	0.12[16]	NO[5,9]
Endosulfan-I	ND	NP	ND	NP	0.067	NA	NO[9]
Endrin	ND	NP	ND	NP	0.049	0.025[16]	NO[5,9]
alpha-Chlordane	ND	NP	ND	NP	0.014	0.50[16]	NO[5]
Chloromethane	ND	NP	0.003	0.55[13]	ND	NP	NO[5]

TABLE 43
Selection of Chemicals for Evaluation in the Ecological Assessment[1]
(all concentrations are in ppm)

Chemical	Sediment[1a]		Water		Fish Tissue		Selected for Evaluation
	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	

1 Maximum detected concentration listed for each environmental medium.

1a Most of the sediment screening values were calculated using the following equation from the USEPA Interim Sediment Criteria Document (USEPA 1988): (AWQC) (K_{oc}) (0.05); where AWQC = ambient water quality criteria, K_{oc}=soil sorption constant, and 0.05 represents the assumption of 5% organic carbon in the sediment. If Ohio EPA water quality standards were found for any chemical, it was used instead of the AWQC. K_{oc} values were obtained from one of the following: Verschueren (1983), Howard et al. (1990), or HSDB (1991). If K_{oc}'s were not available, they were estimated using the K_{ow} and the equation presented in USEPA (1988): $\log K_{oc} = 0.00028 + 0.983 \log K_{ow}$.

2 ND = Not detected.

3 Sediment value developed by NYSDEC (1989).

4 NA = Not available.

5 Observed maximum value is less than the screening value in at least one medium.

6 Toxicity screening value not available for one or more media.

7 Based on Ohio EPA water quality standards. Sediment criterion calculated using the equilibrium partitioning equation found in USEPA Interim Sediment Criteria Document (USEPA 1988).

8 Because no data were available to derive a screening value for 2-methylnaphthalene, the range of values estimated for other polyaromatic hydrocarbons (PAHs) are used.

TABLE 43
Selection of Chemicals for Evaluation in the Ecological Assessment[1]
(all concentrations are in ppm)

Chemical	Sediment[1a]		Water		Fish Tissue		Selected for Evaluation
	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	Maximum Concentration	Toxicity Screening Value	

[9] Frequency of detection is less than 5%.
[10] Ambient Water Quality Criteria (USEPA).
[11] Photomirex concentrations will be summed with mirex concentrations and included in the assessment.
[12] Toxicity value for LC_{50} in fathead minnows (as reported in Verschueren 1983) was used because there were no Ohio or USEPA criteria. It was divided by a safety factor of 1000 (10 for laboratory to field, 10 for acute to chronic, and 10 for difference in species).
[13] USEPA Interim Sediment Criterion (USEPA 1988).
[14] NP = Not applicable.
[15] Derived using the method described in footnote (1a) above.
[16] Taken from Newell et al. 1987.
[17] Derived in Section E of this report (Chapter IX: Off-site Ecological Risk Assessment).

TABLE 44
Mean Mirex Concentrations (μg/kg) in Sediment, Flood Plain Soil and Fish Tissue from Off-Site Sample Stations along MFLBC

Sample Medium	Stations 1-23 (Reach 1)	Stations 24-39 (Reach 2)	Stations 40-52 (Reach 3)	OVERALL AVERAGE
Sediment	244	78.0	23.6	144
Flood Plain Soil	699	368	23.3	555
Fish Tissue	1,663	171	50.1	767

TABLE 45
Selected Repeat-Dose Toxicity Studies of Mirex in Birds

Reference	Species	Dose/Duration	Criteria Evaluated	NOAEL and LOAEL	Reported Findings
Hyde (1972)	Mallard (5F, 2M per pen with 6 replications at each dose level)	Fed 0, 1, or 100 ppm in diet for 25 weeks	Egg production, egg shell thickness, shell weight, embryonation, hatchability, 14-day post-hatch duckling mortality	Decreased survival at 100 ppm LOAEL=100 ppm NOAEL=1 ppm	No effects on egg production, eggshell thickness, shell weight, embryonation (fertility) or hatchability. Decreased survival of ducklings at 100 pm. Mortality at 0, 1, and 100 ppm was 96%, 94%, and 73%, respectively.
Naber and Ware (1965)	Domestic chicken (15 hens per treatment group, 12 hens in control group)	Fed 0, 300 or 600 ppm in diet for 16 weeks	Feed consumption, egg production, body weight changes, hatchability, fertility, embryonic mortality, and chick growth and mortality	LOAEL=300 ppm	No effects on egg production or feed intake. Reduced hen body weight at 300 and 600 ppm. Dose-related reduction in hatchability and chick survival after 6 and 12 weeks on the experimental diet; effects were statistically significant only at the 600 ppm level.
Ivie et al. (1974) (in Waters et al. 1977)	Domestic chicken	Not reported	Not reported	NOAEL=200 ppm	Diets containing up to 200 ppm were tolerated without adverse effects on various reproductive variables, including egg hatching and chick growth and survival.

TABLE 45
Selected Repeat-Dose Toxicity Studies of Mirex in Birds

Reference	Species	Dose/Duration	Criteria Evaluated	NOAEL and LOAEL	Reported Findings
Davison and Cox (1974) and Davison et al. (1975) (in Waters et al. 1977)	Quail (2 species)	Not reported	Not reported	NOAEL=80 ppm	No effects on reproduction in quail fed 80 ppm for 12 weeks.
Kendall at al. (1978)	Bobwhite quail (5-7 breeding pairs per treatment group)	Fed 0, 1, 20, or 40 ppm for 2 generations (F_0 and F_1). F_1 data incomplete for control and 20 and 40 ppm groups.	Hatchability, fertility, chick survival	NOAEL=40 ppm	No adverse effects on mortality or reproduction. F_1 data limited because control and two treatment groups (20 and 40 ppm) were lost at 16 weeks due to predation by dogs. F_1 data for the 1 ppm group showed no reproductive problems; F_1 generation mirex-treated birds laid fertile eggs.

TABLE 46
Selected Repeat-dose Toxicity Studies of Mirex in Mammalian Species

Reference	Species	Dose/Duration	Criteria Evaluated	NOAEL and LOAEL	Reported Findings
Chu et al. (1981)	Sprague-Dawley rats (15M, 20F)	Fed 0, 5, 10, 20, or 40 ppm for 13 weeks before mating, during a 2-week mating period, and through gestation and lactation	Fertility, litter size, gestational survival, 4-day survival, 21-day survival, body weight, adult and pup histopathology	Decreased litter size at 5 ppm LOAEL=5 ppm (0.5 mg/kg/day based on IRIS 1992)	40 ppm females had weight loss, litter size decreased in all groups, survival declined in 20 and 40 ppm groups.
Gaines and Kimbrough (1970)	Sherman rats (10M, 10F per group)	Fed 0, 1, 5, 25 ppm for 166 days	Liver effects	5 ppm (0.2-0.48 mg/kg/day) showed some liver effects. NOAEL=1 ppm (0.04-0.09 mg/kg/day) LOAEL=5 ppm (0.21-0.48 mg/kg/day)	Some liver effects noted in both sexes at 5 ppm, significant effects noted in both sexes at 25 ppm.

TABLE 46
Selected Repeat-dose Toxicity Studies of Mirex in Mammalian Species

Reference	Species	Dose/Duration	Criteria Evaluated	NOAEL and LOAEL	Reported Findings
Larson et al. (1979)	Charles River rats (10M, 10F per group)	Fed 0, 5, 20, 80, 320 and 1280 ppm for 13 weeks	Body weight, mortality, hemoglobin, white cell count, liver effects	Males showed enlarged livers at 80 ppm and both sexes had liver-cell changes at that level LOAEL=80 ppm NOAEL=20 ppm	Mortality at 1280 ppm diet, growth was depressed in both sexes at 1280 ppm and 320 ppm in males, hemoglobin depressed at 320 and 1280 ppm in both sexes, total white cells doubled in 1280 ppm males, enlarged livers in 80 ppm males and above and in females at 320 ppm, liver-cell changes at 80 ppm and above.

TABLE 46
Selected Repeat-dose Toxicity Studies of Mirex in Mammalian Species

Reference	Species	Dose/Duration	Criteria Evaluated	NOAEL and LOAEL	Reported Findings
NTP (1990)	First study: F344/N rats (52F, 52M per group) Second study: F344/N rats (52F per group)	First study: Fed 0, 0.1, 1, 10, 25, or 50 ppm for 104 weeks Second study: Fed 0, 50, 100 ppm for 104 weeks	Liver, adrenal, hematological, and thyroid-gland effects	10 ppm showed liver lesions in both sexes and neoplastic nodules in males (0.1 ppm males showed minimal liver lesions, histological evidence of scattered cellular enlargement not seen in 1 ppm groups, no other toxic effects reported for experimental animals at < 10 ppm) LOAEL=10 ppm (0.7 mg/kg/day) NOAEL=1 ppm (0.07 mg/kg/day)	First study: Male survival decreased at 25 and 50 ppm, liver lesions at 10, 25, 50 and 100 ppm for both sexes, neoplastic nodules in males at 10, 25, and 50 ppm, adrenal-gland lesions at 25 and 50 ppm for males and at 50 ppm for females, kidney lesions at 50 ppm in males, mononuclear cell leukemia in 25 ppm males and females and in 50 ppm females, thyroid-gland lesions at 50 ppm for both sexes. Second study: Neoplastic nodules at 50 and 100 ppm, mononuclear cell leukemia at 25 ppm.

TABLE 46
Selected Repeat-dose Toxicity Studies of Mirex in Mammalian Species

Reference	Species	Dose/Duration	Criteria Evaluated	NOAEL and LOAEL	Reported Findings
Larson et al. (1979)	Beagle dogs (2F, 2M per group)	Fed 0, 4, 20, and 100 ppm for 13 weeks	Mortality, weight gain, and hematological, liver and spleen effects	Dogs at 100 ppm (2.5 mg/kg/day) experienced mortality, decreased weight gain, hematological effects, liver and spleen effects LOAEL=100 ppm (2.5 mg/kg/day) NOAEL=20 ppm (0.5 mg/kg/day)	Decreased weight gain at 100 ppm, mortality in 100 ppm group, elevated hematocrit and WBC counts in 100 ppm male, smaller spleens and enlarged livers in 100 ppm group.
Wolfe et al. (1979) (in Eisler 1985)	Old field mouse (F_1 generation of wild parents), (12 M, 12F per group)	Fed 0, 1.8, or 17.8 ppm (0, 0.24, or 2.4 mg/kg/day) for 15 months	Mortality, number litters produced, litter size (litters evaluated at 3-month intervals)	Study not adequate for determination of NOAEL and LOAEL.	Significant mortality was seen in the mice at the high dose (17.8 ppm) group. Reproduction essentially stopped in 17.8 ppm group after 3 months. Authors noted suggestion of decreased reproduction at 1.8 ppm mirex (primarily decrease in litter size). Statistical analysis, however, was not performed. Investigators noted that other studies have shown that effects on litter size may not be consistent.

TABLE 47 Estimated Mean Daily Mirex Doses for Receptor Species			
Receptor Species	Estimated Mean Daily Dose by MFLBC Reach (μg/kg/day)		
	Reach 1-23	Reach 24-39	Reach 40-52
Heron	333.0	34.3	10.1
Kingfisher	831.5	85.3	25.1
Sora	418.5	133.6	40.4
Virginia rail	432.7	138.1	41.8
Robin	47.8	25.2	1.6
Northern harrier	11.7	6.2	0.4
Red fox	5.8	3.1	0.2
Mink	153.8	25.9	7.2

TABLE 48
Comparisons of Adverse Effects Thresholds With Estimated Daily Doses

Indicator Species	Adverse effect threshold (μg/kg/d)	Estimated Daily Dose by MFLBC Reach (μg/kg/d)			Ratio of Estimated Daily Exposure to Adverse Effect Threshold[a]		
		Reach 1-23	Reach 24-39	Reach 40-52	Reach 1-23	Reach 24-39	Reach 40-52
Heron	3,600	333.0	34.3	10.1	0.093	0.010	0.003
Kingfisher	3,600	831.5	85.3	25.1	0.232	0.024	0.007
Sora	3,600	418.5	133.6	40.4	0.120	0.037	0.011
Virginia rail	3,600	432.7	139.1	41.8	0.120	0.039	0.012
Northern harrier	3,600	11.7	6.2	0.4	0.003	0.002	<0.001
American robin	3,600	47.8	25.2	1.6	0.011	0.007	<0.001
Red fox	100	5.8	3.1	0.2	0.058	0.031	0.002
Mink	100	153.8	25.9	7.2	1.538	0.254	0.072

[a] ratios greater than 1 indicate that the exposure exceeds the adverse effects threshold and a risk is consequently predicted for the indicator species.

TABLE 49 Comparison of Mean Sediment Mirex Concentration With Sediment Quality Criteria		
Sample Station	Mirex (μg/kg)	Ratio of sediment concentration to sediment criteria 1,200 μg/kg @ 5% oc[a]
1	22.7	0.02
2	17.7	0.01
3	24.7	0.02
4	25.9	0.02
5	155.8	0.13
6a	83.0	0.07
6b	32.9	0.03
6c	100.4	0.08
6d	136.5	0.11
7	263.9	0.22
10	1687.4	1.40
11	568.0	0.47
13	558.1	0.47
14	1202.3	1.00
15	152.6	0.13
16	46.9	0.04
17	72.8	0.06
18	70.4	0.06
19	137.2	0.11
19a	39.3	0.03
19b	122.3	0.10
21	46.0	0.04
22	187.8	0.16
23	110.0	0.09

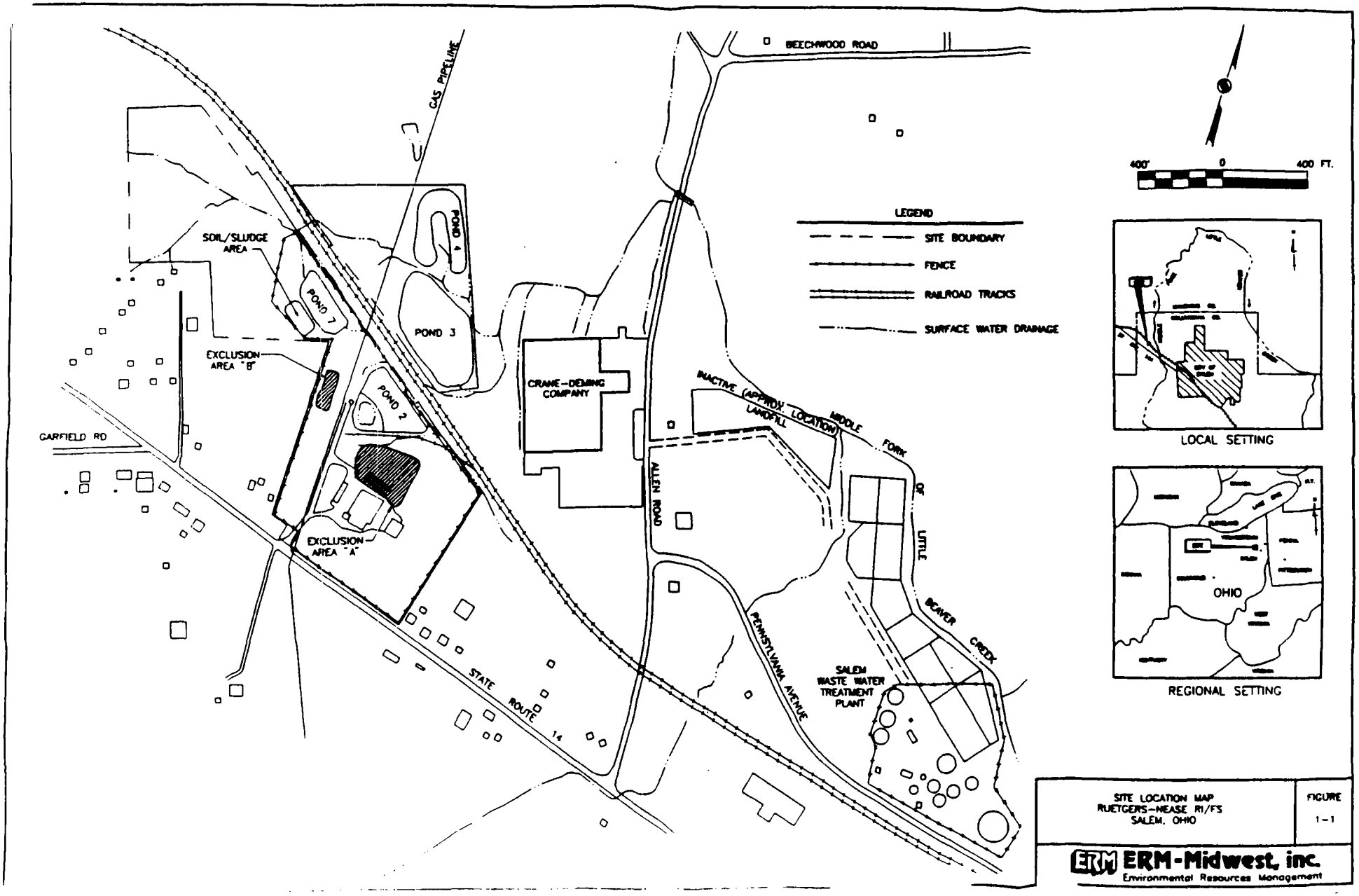
TABLE 49
Comparison of Mean Sediment Mirex Concentration With Sediment Quality Criteria

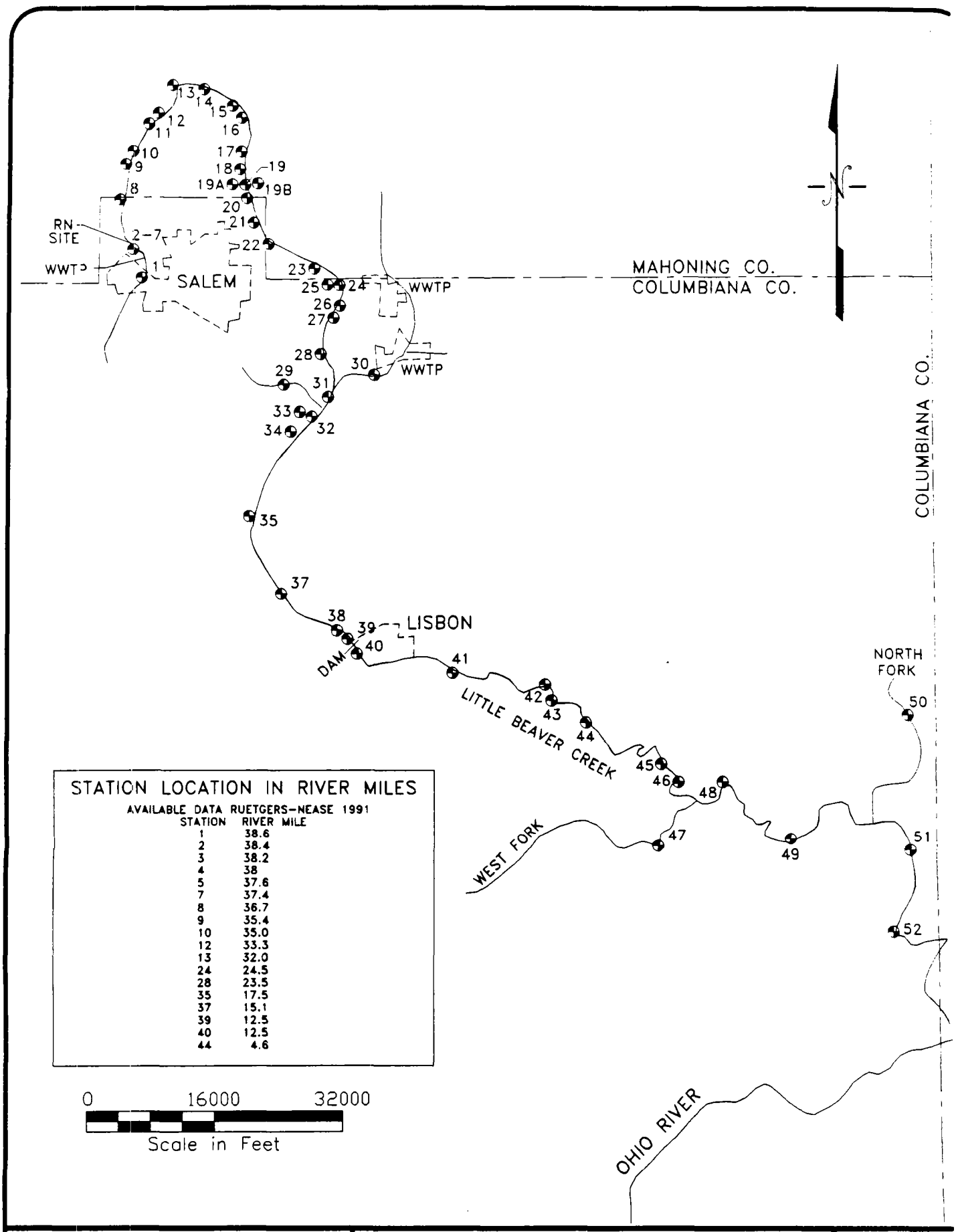
Sample Station	Mirex (μg/kg)	Ratio of sediment concentration to sediment criteria 1,200 μg/kg @ 5% oc[a]
24	141.0	0.12
25	90.8	0.08
26	194.9	0.16
27	171.3	0.14
29	25.1	0.02
31	56.8	0.05
32	47.9	0.04
33	90.2	0.08
34	30.6	0.03
35	25.1	0.02
37	38.6	0.03
38	71.6	0.06
39	30.0	0.02
40	25.5	0.02
41	24.9	0.02
42	22.4	0.02
43	29.2	0.02
44	18.6	0.02
45	23.2	0.02
46	23.9	0.02
47	22.8	0.02
48	23.5	0.02
49	23.0	0.02
50	23.7	0.02
51	23.0	0.02

TABLE 49
Comparison of Mean Sediment Mirex Concentration With Sediment Quality Criteria

Sample Station	Mirex (μg/kg)	Ratio of sediment concentration to sediment criteria 1,200 μg/kg @ 5% oc[a]
52	23.0	0.02

[a] Ratios of sediment concentration to estimated sediment criteria that exceed 1 indicate a potential risk to benthic organisms.


BEECHWOOD ROAD
GAS PIPELINE
POND 4
POND 3
POND 7
POND 2
SOIL/SLUDGE AREA
EXCLUSION AREA "B"
EXCLUSION AREA "A"
GARFIELD RD
CRANE-DEMING COMPANY
INACTIVE (APPROX. LOCATION) LANDFILL
ALLEN ROAD
MIDDLE FORK OF LITTLE BEAVER CREEK
PENNSYLVANIA AVENUE
STATE ROUTE 14
SALEM WASTE WATER TREATMENT PLANT
LEGEND
SITE BOUNDARY
FENCE
RAILROAD TRACKS
SURFACE WATER DRAINAGE
400' 0 400 FT.
LOCAL SETTING
OHIO
REGIONAL SETTING
SITE LOCATION MAP
RUETGERS-NEASE RI/FS
SALEM, OHIO
FIGURE
1-1
ERM-Midwest, inc.
Environmental Resources Management



STATION LOCATION IN RIVER MILES

AVAILABLE DATA RUETGERS-NEASE 1991

STATION	RIVER MILE
1	38.6
2	38.4
3	38.2
4	38
5	37.6
7	37.4
8	36.7
9	35.4
10	35.0
12	33.3
13	32.0
24	24.5
28	23.5
35	17.5
37	15.1
39	12.5
40	12.5
44	4.6

0 16000 32000

Scale in Feet

c:\acad\root\8x11base

ENVIRON

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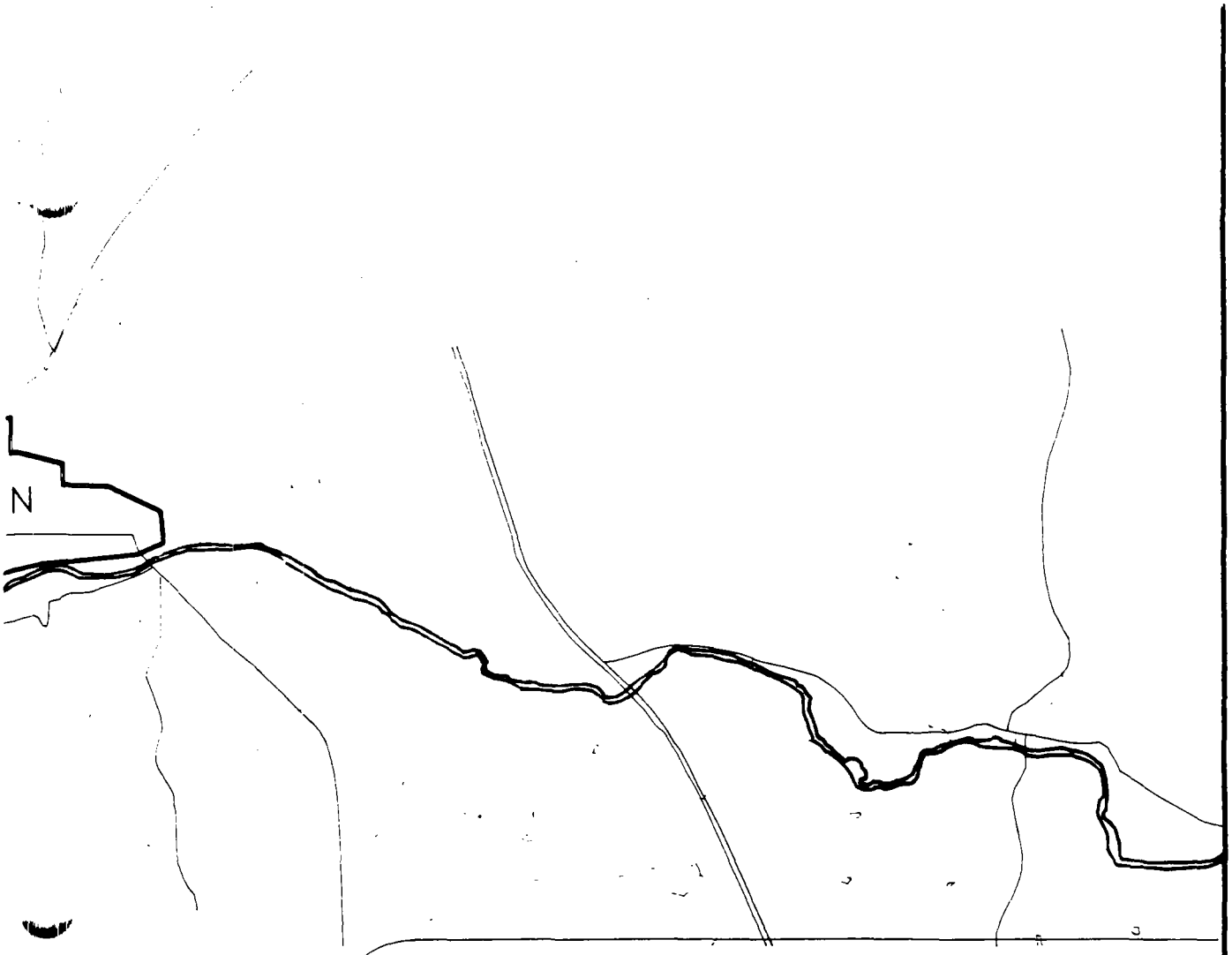
LOCATIONS OF SAMPLING STATIONS

Figure 2



ENVIRON

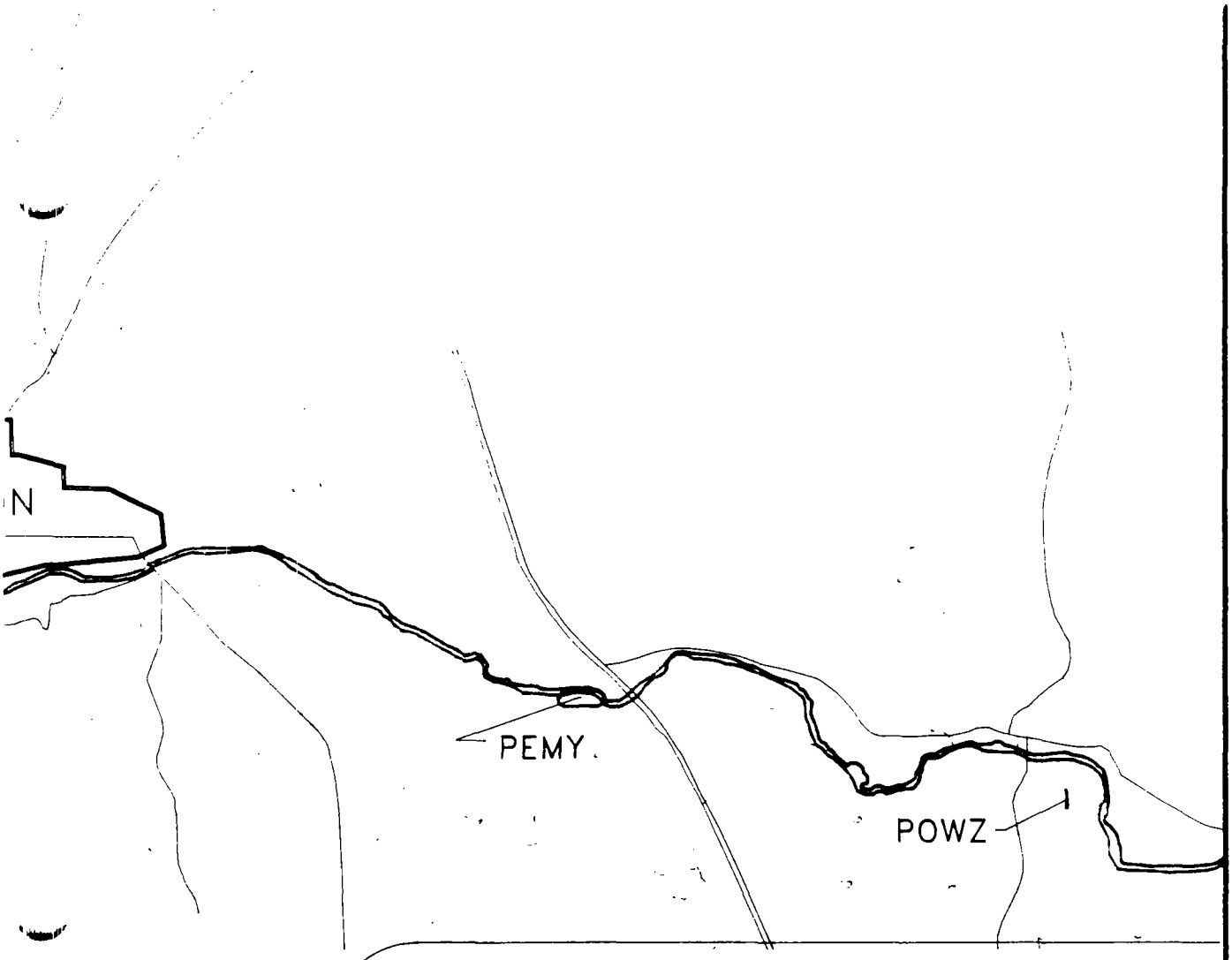
4350 FAIRFAX DR., ARLINGTON, VA 22203

PLATE: 3

WATERSHED OF MFLBC WITH FEATURES WITHIN 1 MILE OF CREEK

SOURCE: U.S.G.S 7.5 minute topographic quadrangles; Elkton, Salem, Lisbon and Damascus, Ohio

DATE: 07/01/93	DRAFTED BY: RGM CHECKED BY: EO
C:\ACADDATA\010439D\BASEMAP	



ENVIRON

4350 FAIRFAX DR., ARLINGTON, VA 22203

PLATE: 4

WETLANDS WITHIN 1 MILE OF MFLBC

SOURCE: U.S. Department of the Interior, National Wetlands Inventory; Elkton, Salem, Lisbon and Damascus, Ohio Quadrangles

DATE: 07/01/93	DRAFTED BY: RGM CHECKED BY: EO
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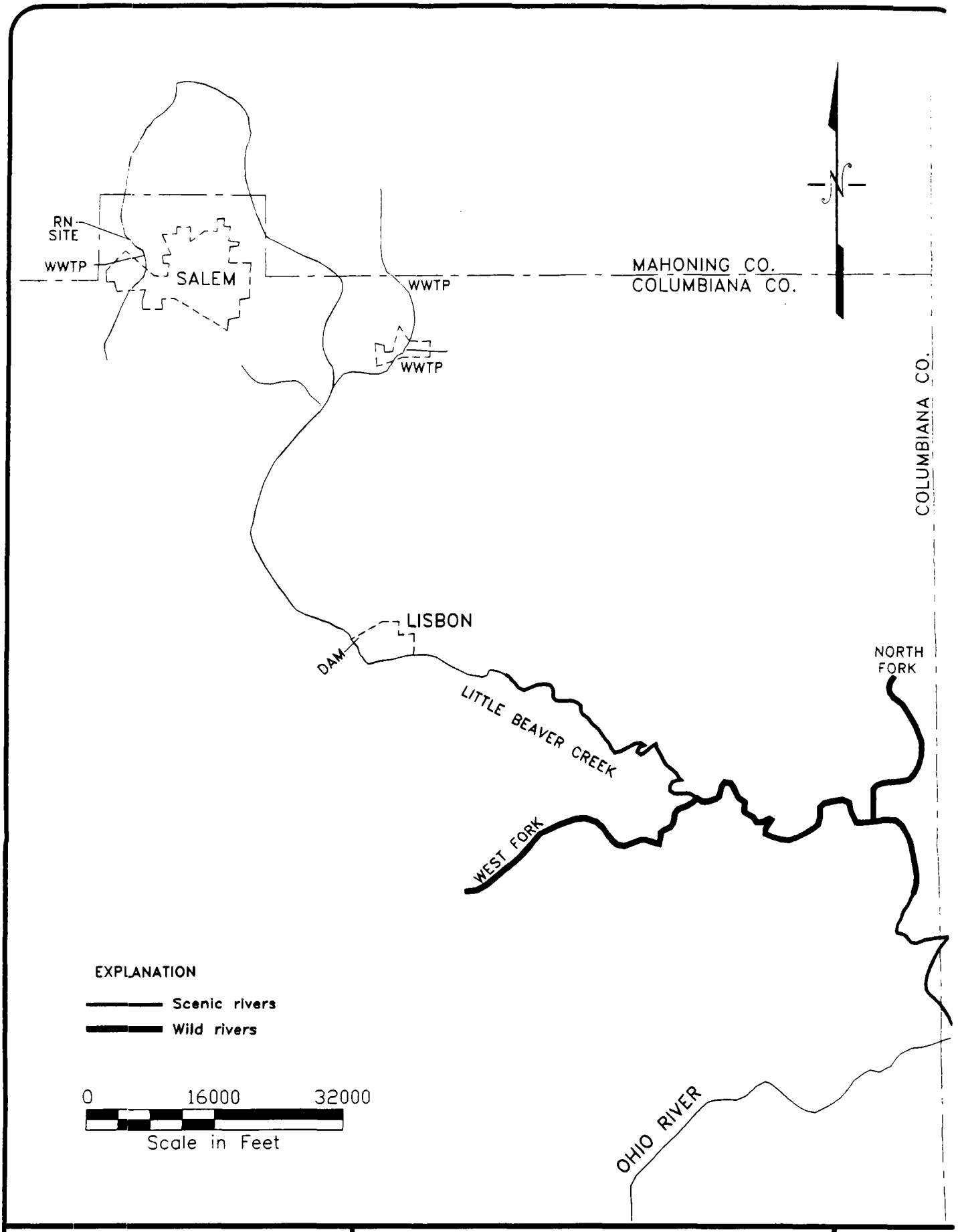
WATER/
Bottom



c:\acad\root\WLDRIVER

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LOCATION OF WILD AND SCENIC RIVERS

Figure 5

ENVIRON

4350 FAIRFAX DR., ARLINGTON, VA 22203

PLATE: 6

FORESTED & OPEN AREAS IMMEDIATELY ADJACENT TO THE MFLBC RIPARIAN ZONE

DATE: 07/01/93	DRAFTED BY: RGM CHECKED BY: EO
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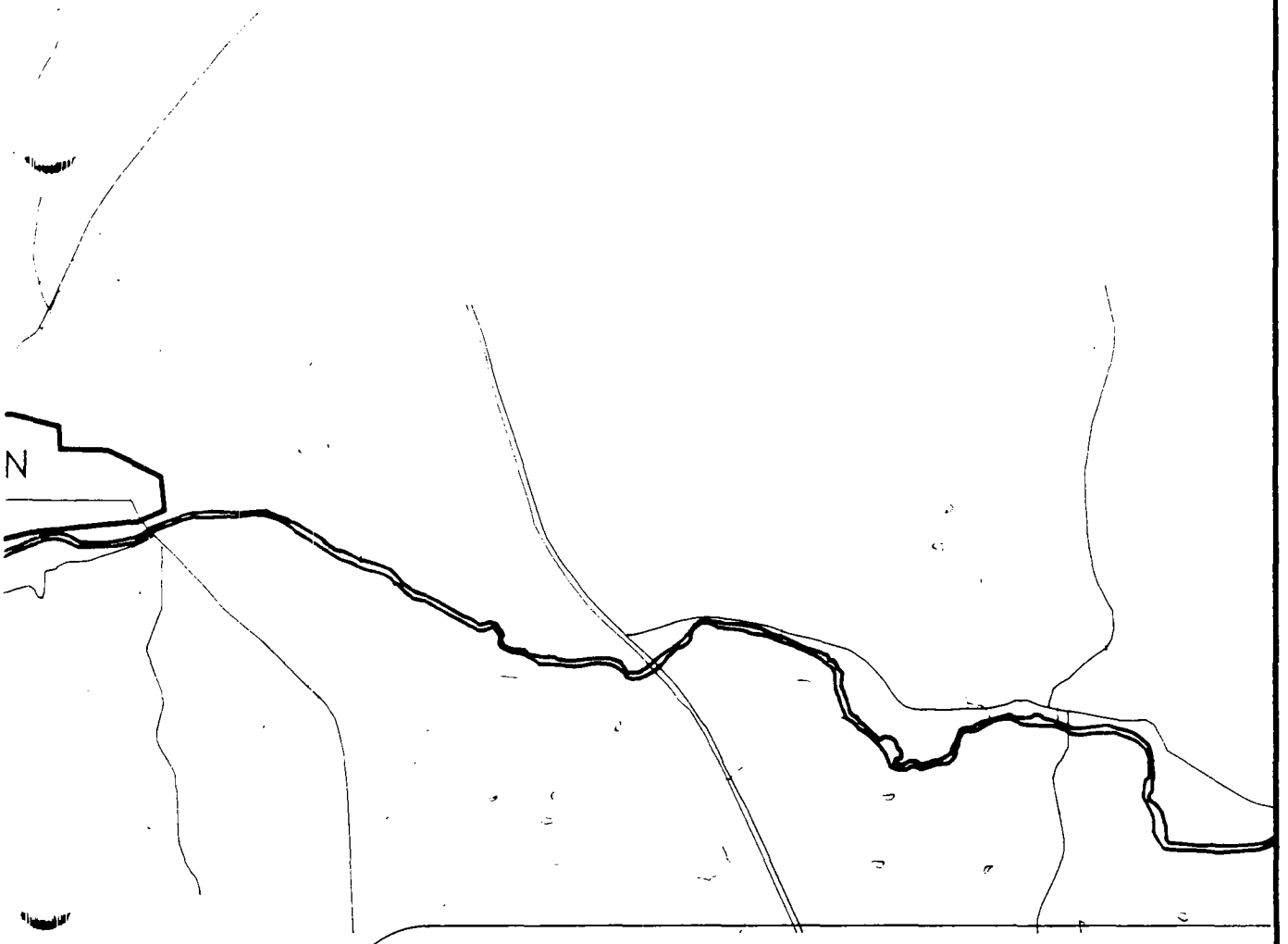

ENVIRON

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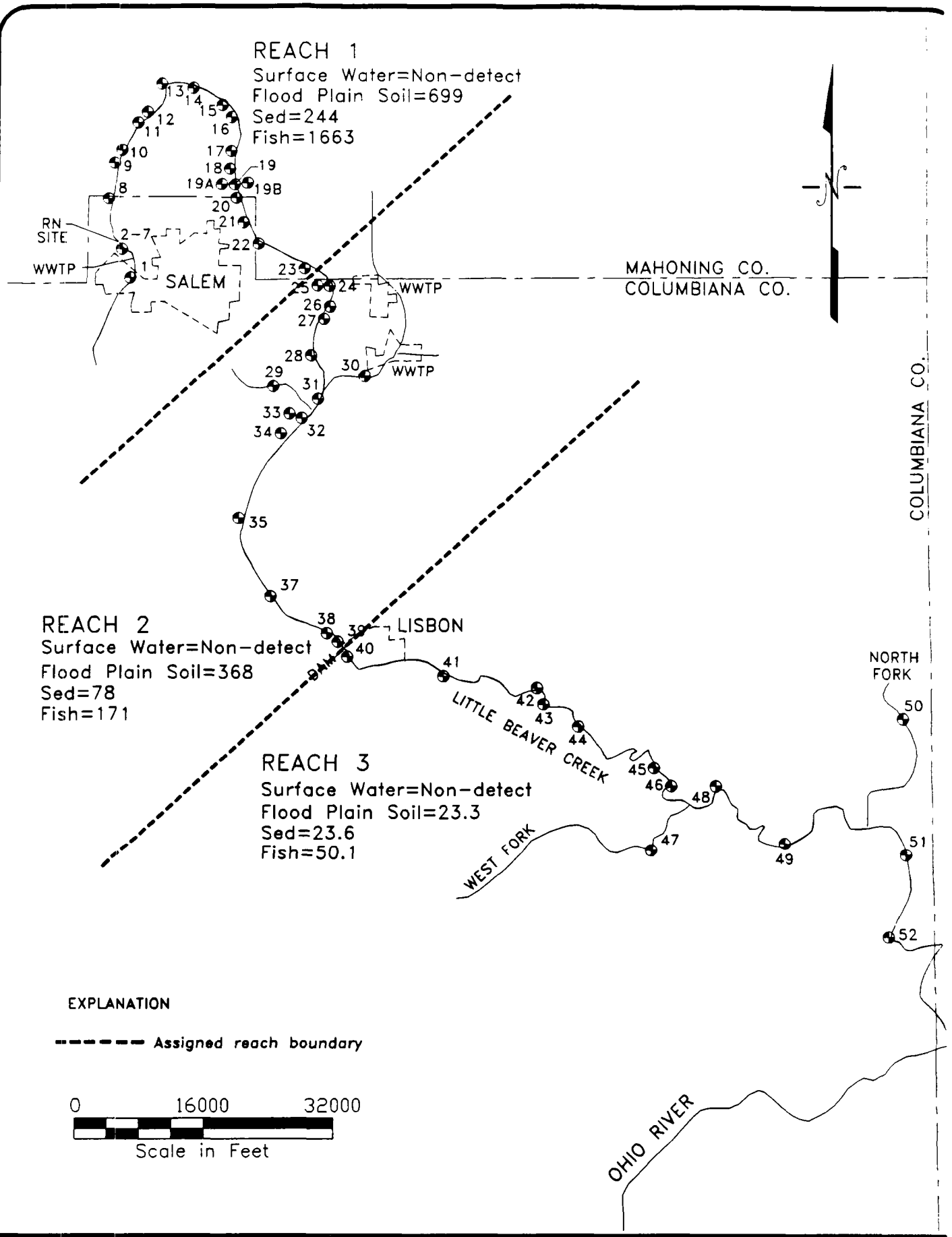
PLATE: 7

LOCATIONS OF THREATENED, ENDANGERED AND SPECIAL CONCERN SPECIES WITHIN 1.5 MILES OF MFLBC

SOURCE: Ohio Department of Natural Resources, Division of Natural Areas and Preserves, Natural Heritage Data Services

DATE: 07/01/93	DRAFTED BY: RGM CHECKED BY: EO
C:\ACADDATA\010439D\BASEMAP	



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ENVIRON
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ASSIGNED REACHES OF MFLBC
FOR USE IN ECOLOGICAL RISK ASSESSMENT
(MEAN MIREX CONCENTRATIONS
BY MEDIUM IN ug/Kg)

Figure
8

APPENDIX A

Summary of Tables of Toxicity Values

INTRODUCTION

Tables A-1 and A-2 in this appendix summarize chronic oral Reference Doses, RfDs, for the noncarcinogenic effects of chemicals and cancer slope factors for carcinogens for all chemicals detected in soils, sediment, ground water and surface water at the Site. The toxicity values presented in these tables were used both in the concentration and toxicity screen (see Chapter III) and in the quantitative risk assessment. Tables A-3 and A-4 summarize inhalation RfCs and unit risks for the chemicals detected in air. The RfC and unit risk values were used in the concentration and toxicity screen and in the quantitative risk assessment.

Where available, USEPA-derived toxicity values were summarized in the tables that follow and were used in the risk assessment for the Site. Independent toxicity assessments were performed for three chemicals—mirex, photomirex, and kepone—for which USEPA has not derived toxicity values. Toxicity values developed for mirex, photomirex, and kepone as part of these assessments are summarized in this appendix; toxicological profiles that provided a detailed basis for these values are presented in Appendices D, E, and F.

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Volatile Compounds						
1,1-Dichloroethene	9×10^{-3}	50 ppm in drinking water (9 mg/kg/day) for 2 years.	Rat	Hepatic lesions	1,000 (M)	IRIS 1993
1,1,1-Trichloroethane	9×10^{-2}	500 ppm (NOAEL) for 6 mos	Guinea pigs	Liver toxicity	1,000	HEAST 1992
1,1,2-Trichloroethane	4×10^{-3}	20 mg/L in drinking water (3.9 mg/kg/day)	Mouse	Clinical serum chemistry	1,000	IRIS 1993
1,1,2,2-Tetrachloroethane	NA					
1,2-Dichloroethane	NA					
1,2-Dichloroethene (mixed isomers)	9×10^{-3}	50 ppm in drinking water for 2 years	Rat	Liver lesions	1,000	HEAST 1992
1,2-Dichloropropane	NA					
2-Butanone (methyl ethyl ketone)	6×10^{-1}	1,771 mg/kg/day by drinking water (multigenerational)	Rat	Decreased fetal birth weights	3,000 (L)	IRIS 1993
Acetone	1×10^{-1}	100 mg/kg/day orally for subchronic duration	Rat	NOEL	1,000	IRIS 1993

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Benzene	NA					
Bromoform	2×10^{-2}	25 mg/kg/day by gavage, 5 days/wk for 12 wks	Rat	Hepatic lesions	1,000 (M)	IRIS 1993
Carbon disulfide	1×10^{-1}	20 ppm (11.0 mg/kg/day) by inhalation 34 weeks before breeding plus length of pregnancy	Rat, Rabbit	Fetal toxicity and malformations	100 (M)	IRIS 1993
Carbon tetrachloride	7×10^{-4}	1 mg/kg/day by gavage, 5 days/wk for 12 weeks	Rat	Hepatic lesions	1,000 (M)	IRIS 1993
Chlorobenzene	2×10^{-2}	27.25 mg/kg/day by capsule, 5 days/wk for 13 weeks	Dog	Histopathologic changes in liver	1,000 (M)	IRIS 1993
Chloroethane	3×10^{0} (converted from chronic RfC[a])	1,504 ppm (NOAEL) by intermittent inhalation for 10 days	Mouse	Delayed ossification of fetus	300	HEAST 1992
Chloroform	1×10^{-2}	15 mg/kg/day by capsule for 6 days/week for 7.5 years	Dog	Fatty cyst formation in liver	1,000 (M)	IRIS 1993
Chloromethane	NA					

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Dibromochloromethane	2×10^{-2}	30 mg/kg/day by gavage, 5 days/week for 13 weeks	Rat	Hepatic lesions	1,000 (M)	IRIS 1993
Ethylbenzene	1×10^{-1}	136 mg/kg/day by gavage, 5 days/wk for 182 days	Rat	Liver and kidney toxicity	1,000 (L)	IRIS 1993
Methylene chloride	6×10^{-2}	5.85(males) and 6.47(females) mg/kg/day in drinking water for 2 years	Rat	Liver toxicity	100 (M)	IRIS 1993
Styrene	2×10^{-1}	200 mg/kg/day by gavage	Dog	liver and red blood cell effects	1,000 (M)	IRIS 1993
Tetrachloroethene	1×10^{-2}	20 mg/kg/day by gavage, 5 days/wk for 6 wks	Mouse, rat	Hepatoxicity (mouse), weight gain (rat)	1,000 (M)	IRIS 1993
Toluene	2×10^{-1}	223 mg/kg/day by gavage for 13 wks	Rat	Liver and kidney weight changes	1,000 (M)	IRIS 1993
Trichloroethene	NA					
Vinyl Chloride	NA					

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Xylene (total)	$2 \times 10^{+0}$	250 mg/kg/day by gavage 5 days/week for 103 weeks	Rat	increased mortality, hyperactivity, deceased body weight	100 (M)	IRIS 1993
Semivolatile Compounds						
1,2-Dichlorobenzene	9×10^{-2}	120 mg/kg/day by gavage for 2 years	Rat	NOAEL	1,000 (L)	IRIS 1993
1,2,4-Trichlorobenzene	1×10^{-2}	14.8 mg/kg/day (multigenerational)	Rat	Vacuolization of zona fasciculata in cortex, increased adrenal weight	1,000 (M)	IRIS 1993
1,3-Dichlorobenzene	NA					
1,4-Dichlorobenzene	2×10^{-1} (converted from chronic RfC*)	454.6 mg/m^3 (NOAEL) by intermittent inhalation for 76 weeks	Rat	Liver and kidney effects	100	HEAST 1992
2-Chloronaphthalene	8×10^{-2}	250 mg/kg/day by gavage for 13 weeks	Mouse	abnormal appearance, liver enlargement, dysnea	3,000 (L)	IRIS 1993
2-Chlorophenol	5×10^{-3}	50 ppm (5 mg/kg/day) in drinking water	Rat	Reproductive effects	1,000 (L)	IRIS 1993

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
2-Methylnaphthalene	NA					
2-Methylphenol (o-cresol)	5×10^{-2}	50 mg/kg/day (NOAEL); 100 mg/kg/day (LOAEL)	Rat	Decreased body weight and neurotoxicity	1,000 (M)	IRIS 1993
2-Nitroaniline (o-)	6×10^{-5}	9.8 mg/m^3 (LOAEL); intermittent inhalation for 4 weeks	Rat	Hematological effects	10,000	HEAST 1992
2-Nitrophenol	NA					
2,4-Dichlorophenol	3×10^{-3}	0.3 mg/kg/day in drinking water for 90 days, then 15 weeks	Rat	Decreased delayed hypersensitivity repsonse	100 (L)	IRIS 1993
2,4-Dimethylphenol	2×10^{-2}	50 mg/kg/day by gavage for 90 days	Mouse	hematological changes, lethargy, prostration, ataxia	3,000 (L)	IRIS 1993
2,4-Dinitrotoluene	2×10^{-3}	0.2 mg/kg/day in diet for 2 years	Dog	Neurotoxicity, hyperplasia of biliary tract and Heinz bodies	100 (H)	IRIS 1993
2,4,6-Trichlorophenol	NA					

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
2,6-Dinitrotoluene	NA					
3-Nitroaniline (m-)	NA					
3,4-Dichloronitrobenzene	NA					
4-Chloro-3-methylphenol	NA					
4-Chloroaniline	4×10^{-3}	12.5 mg/kg/day in diet for 78 weeks	Rat	Nonneoplastic lesions of the splenic capsule	3,000 (L)	IRIS 1993
4-Chlorophenyl-phenylether	NA					
4-Methylphenol (p-cresol)	5×10^{-3}	5 mg/kg/day (NOEL); gavage	Rabbit	maternal death, respiratory distress, eye discharge, cyanosis, hypoactivity	1,000	HEAST 1992
4-Nitroaniline (p-)	NA					
Acenaphthene	6×10^{-2}	175 mg/kg/day by gavage for 90 days	Mouse	Hepatoxicity	3,000 (L)	IRIS 1993
Acenaphthylene	NA					
Anthracene	3×10^{-1}	1,000 mg/kg/day by gavage for 90 days	Mouse	no effects	3,000 (L)	IRIS 1993

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Benzoic acid	4×10^{0}	34 mg/day (daily per capita intake)	Human	NOAEL	1 (M)	IRIS 1993
Benzo(a)anthracene	NA					
Benzo(a)pyrene	NA					
Benzo(b)fluoranthene	NA					
Benzo(g,h,i)perylene	NA					
Benzo(k)fluoranthene	NA					
Benzyl alcohol	3×10^{-1}	286 mg/kg/day by gavage for 103 weeks	Rat	Epithelial hyperplasia in forestomach	1,000	HEAST 1992
bis(2-Chloroethoxy)methane	NA					
bis(2-Chloroethyl)ether	NA					
bis(2-Ethylhexyl)phathalate	2×10^{-2}	19 mg/kg bw/day in diet for 1 - 2 years	Guinea pig	Increase in relative liver weights	1,000 (M)	IRIS 1993
Butylbenzylphthalate	2×10^{-1}	2,800 ppm (159 mg/kg/day) in diet for 26 weeks	Rat	Increase liver to body weight and liver to brain ratios	1,000 (L)	IRIS 1993
Carbazole	NA					

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Chrysene	NA					
Dibenzofuran	NA					
Diethylphthalate	8×10^{-1}	750 mg/kg bw/day in diet for 16 weeks	Rat	Altered organ weights, decreased growth and food consumption rates	1,000 (L)	IRIS 1993
Dimethyl phthalate	1×10^{1}	1000 mg/kg/day in diet for 2 years	Rat	Kidney effects	100	HEAST 1992
Di-n-butylphthalate	1×10^{-1}	125 mg/kg/day in diet for 1 year	Rat	Increased mortality	1,000 (L)	IRIS 1993
Di-n-octylphthalate	2×10^{-2}	175 mg/kg/day, 7-12 months in diet	Rat	Increased liver and kidney weight, increased liver serum	1,000	HEAST 1992
Diphenylsulfone	NA					
Fluoranthene	4×10^{-2}	125 mg/kg/day by gavage for 13 weeks	Mouse	nephropathy, increased liver weights, hematological alterations	3,000 (L)	IRIS 1993

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Fluorene	4×10^{-2}	125 mg/kg/day by gavage for 13 weeks	Mouse	decreased RBC, hemoglobin, and packed cell volume	3,000 (L)	IRIS 1993
Hexachlorobenzene	8×10^{-4}	1.6 ppm in diet (0.08 mg/kg/day) for 130 wks	Rat	Liver effects	100 (M)	IRIS 1993
Hexachlorobutadiene	2×10^{-4}	0.2 mg/kg/day in diet for 2 years	Rat	Kidney toxcity	100 (L)	IRIS 1993
Hexachlorocyclopentadiene	7×10^{-3}	7 mg/kg/day by gavage for 13 weeks	Rat	Stomach lesions	1,000 (L)	IRIS 1993
Hexachloroethane	1×10^{-3}	1 mg/kg/day in diet for 16 weeks	Rat	Renal tubules atrophy and degeneration	1,000 (M)	IRIS 1993
Indeno(1,2,3-cd)pyrene	NA					
Naphthalene	4×10^{-2}	50 mg/kg/day, 5 days/week for 13 weeks	Rat	NOEL	1,000	USEPA/ ECAO 1993
Nitrobenzene	5×10^{-4}	4.6 mg/kg/day by inhalation for 90 days	Rat, mouse	hepatic, renal, hematologic, and adrenal lesions	10,,000 (L)	IRIS 1993

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
N-Nitrosodiphenylamine	NA					
N-Nitroso-di-n-propylamine	NA					
Pentachlorophenol	3×10^{-2}	3 mg/kg/day in diet for 2 years	Rat	Pigmentation of liver and kidney	100 (M)	IRIS 1993
Phenanthrene	NA					
Phenol	6×10^{-1}	60 mg/kg/day by gavage on days 6-15 of gestation	Rat	Reduced fetal body weights	100 (L)	IRIS 1993
Pyrene	3×10^{-2}	75 mg/kg/day by gavage for 13 weeks	Mouse	Kidney effects	3,000 (L)	IRIS 1993
Pesticides						
4,4'-DDD	NA					
4,4'-DDE	NA					
4,4'-DDT	5×10^{-4}	1 ppm DDT in diet (0.05 mg/kg bw/day) for 27 weeks	Rat	Liver lesions	100 (M)	IRIS 1993
Aldrin	3×10^{-5}	0.5 ppm Aldrin in diet for 2 years	Rat	Liver toxicity	1,000 (M)	IRIS 1993

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
alpha-BHC	NA					
beta-BHC	NA					
delta-BHC	NA					
Chlordane (alpha-, gamma-)	6×10^{-5}	30 month feeding study	Rat	Regional liver hypertrophy in females	1,000	IRIS 1993
Dieldrin	5×10^{-5}	0.1 ppm Dieldrin in diet (0.005 mg/kg/day) for 2 years	Rat	Liver lesions	100 (M)	IRIS 1993
Endosulfan (I and II)	5×10^{-5}	0.15 mg/kg/day (LOAEL) in diet for 2 generations	Rat	Kidney lesions	3,000	HEAST 1992
Endosulfan Sulfate	NA					
Endrin	3×10^{-4}	1 ppm Endrin in diet (0.025 mg/kg/day) for 2 years	Dog	Occassional convulsions, mild histopathological effects in the liver	100 (M)	IRIS 1993
Endrin aldehyde	NA					

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
gamma-BHC (lindane)	3×10^{-4}	4 ppm in diet (0.33 mg/kg/day) for 12 weeks	Rat	Liver and kidney toxicity	1,000 (M)	IRIS 1993
Heptachlor	5×10^{-4}	3 ppm Heptachlor in diet (0.15 mg/kg/day) for 2 years	Rat	Increased liver to body weight in males	300 (L)	IRIS 1993
Methoxychlor	5×10^{-3}	5.01 mg/kg/day for days 7 - 19 of gestation	Rabbit	Excessive loss of litters	1,000 (L)	IRIS 1993
Other						
Kepone	6.5×10^{-4}	5 ppm for 128 days in diet	Mouse	Reduced litter number and size	1,000	See Appendix F
Mirex	2×10^{-4}	1 ppm Mirex in diet (0.07 mg/kg/day) for 104 weeks	Rat	Liver cytomegaly, fatty metamorphosis,angie ctasis, thyroid cystic follicles	300 (H)	IRIS 1993
Photomirex	1.25×10^{-3}	Diet	Rat	Reduced litter size	100	See Appendix E

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Dioxins and Furans						
Total TCDDs	NA	See TEF scheme at end of table				USEPA 1989
2,3,7,8-TCDD	1×10^{-9}					USEPA 1985
Total PeCDDs	NA	See TEF scheme at end of table				USEPA 1989
Total HxCDDs	NA	See TEF scheme at end of table				USEPA 1989
Total HpCDDS	NA	See TEF scheme at end of table				USEPA 1989
1,2,3,4,6,7,8-HpCDD	NA	See TEF scheme at end of table				USEPA 1989
Total TCDFs	NA	See TEF scheme at end of table				USEPA 1989
2,3,7,8-TCDF	NA	See TEF scheme at end of table				USEPA 1989
OCDD	NA	See TEF scheme at end of table				USEPA 1989

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Total PeCDF	NA	See TEF scheme at end of table				USEPA 1989
1,2,3,7,8-PeCDF	NA	See TEF scheme at end of table				USEPA 1989
2,3,4,7,8-PeCDF	NA	See TEF scheme at end of table				USEPA 1989
Total HxCDF	NA	See TEF scheme at end of table				USEPA 1989
1,2,3,4,7,8-HxCDF	NA	See TEF scheme at end of table				USEPA 1989
1,2,3,6,7,8-HxCDF	NA	See TEF scheme at end of table				USEPA 1989
2,3,4,6,7,8-HxCDF	NA	See TEF scheme at end of table				USEPA 1989
Total HpCDF	NA	See TEF scheme at end of table				USEPA 1989
1,2,3,4,6,7,8-HpCDF	NA	See TEF scheme at end of table				USEPA 1989

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
1,2,3,4,7,8,9-HpCDF	NA	See TEF scheme at end of table				USEPA 1989
OCDF	NA	See TEF scheme at end of table				USEPA 1989
Inorganics						
Aluminum	NA					
Antimony	4×10^{-4}	5 ppm in water for lifetime	Rat	Longevity, blood glucose and cholesterol	1,000 (L)	IRIS 1993
Arsenic	3×10^{-4}	0.0008 mg/kg/day in food and water	Human	Hyperpigmentation, keratosis and possible vascular complications	3 (M)	IRIS 1993
Barium	7×10^{-2}	0.21 mg/kg/day in drinking water	Human	Increased blood pressure	3 (M)	IRIS 1993
Beryllium	5×10^{-3}	5 ppm in drinking water for lifetime	Rat	NOAEL	100 (L)	IRIS 1993
Cadmium	5×10^{-4} (water) 1×10^{-3} (food)	0.005 mg/kg/day in water, 0.01 mg/kg/day in food	Human	Proteinuria	10 (H)	IRIS 1993

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Calcium	ESS. NUTRIENT					
Chromium	5×10^{-3}	25 mg/l in drinking water for 1 year	Rat	No effects	500 (L)	IRIS 1993
Cobalt	NA					
Copper	ESS. NUTRIENT					
Cyanide	2×10^{-2}	10.8 mg/kg/day in diet for 2 years	Rat	weight loss, thyroid effects, myelin degeneration	100 (M)	IRIS 1993
Iron	NA					
Lead	NA					
Magnesium	ESS. NUTRIENT					
Manganese	ESS. NUTRIENT					
Mercury	3×10^{-4}	oral, parenteral	Rat	Kidney effects	1000	HEAST 1992
Nickel	2×10^{-2}	100 ppm in diet for 2 years	Rat	decreased body and organ weights	300 (M)	IRIS 1993
Potassium	ESS. NUTRIENT					

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Selenium	5×10^{-3}	0.015 mg/kg/day lifetime exposure	Human	Clinical selenosis	3 (H)	IRIS 1993
Silver	5×10^{-3}	1 g i.v. 2 to 9 years	Human	Argyria	3 (L)	IRIS 1993
Sodium	ESS. NUTRIENT					
Thallium	NA					
Vanadium	7×10^{-3}	5 ppm in drinking water for a lifetime	Rat	NOAEL	100	HEAST 1992
Zinc	ESS. NUTRIENT					

[a] Based on the chronic Reference Concentration (RfC); converted to units of mg/kg/day using the assumptions of a 20 m^3/day breathing rate and 70 kg body weight.

NA Not Available

ESS NUTRIENT: Chemical not evaluated in risk assessment because it is an essential nutrient.

HEAST: U.S. Environmental Protection Agency (USEPA). Office of Emergency and Remedial Response. Office of Research and Development. 1992. *Health effects assessment summary tables.* Annual: FY 1992; July 1992 and November 1992 Supplemental Updates. Washington, D.C.

IRIS: U.S. Environmental Protection Agency (USEPA). Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office. 1993. *Integrated risk information system (IRIS).* Cincinnati, OH.

TABLE A-1
Chronic Oral Reference Doses (RfDs) for Chemicals Detected in Site Media

Compounds	Chronic RfD (mg/kg/day)	Basis for RfD				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source

<u>Toxicity Equivalence Factor (TEF) Approach for Chlorinated Dibenzo-p-dioxins and Dibenzofurans (CDDs and CDFs)</u>: The TEF procedure was used for CDDs and CDFs. This method relates the toxicity of CDDs and CDFs to 2,3,7,8-TCDD in the following fashion: CDD and CDF congener concentrations are multiplied by the TEFs listed below to express the concentration in terms of 2,3,7,8-TCDD equivalents. The products are summed to obtain the total 2,3,7,8-TCDD equivalents in the sample. The concentration, expressed in terms of 2,3,7,8-TCDD equivalents, is combined with exposure assumptions and the appropriate toxicity value for 2,3,7,8-TCDD to estimate the risk associated with the mixture of CDDs and CDFs.

CDD	TEF	CDF	TEF
Mono-, Di- or TriCDDs	0	Mono-, Di-, TriCDFs	0
2 3 7 8-TCDD	1	2 3 7 8-TCDFs	0.1
Other TCDDs	0	Other TCDFs	0
2 3 7 8-PeCDDs	0.5	1 2 3 7 8-PeCDF	0.05
Other PeCDDs	0	2 3 4 7 8-PeCDFs	0.5
2 3 7 8-HxCDDs	0.1	Other PeCDFs	0
Other HxCDDs	0	2 3 7 8-HxCDFs	0.1
2 3 7 8-HpCDDs	0.01	Other HxCDFs	0
Other HpCDDs	0	2 3 7 8-HpCDFs	0.01
		Other HpCDFs	0
OCDD	0.001	OCDF	0.001

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TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Volatile Compounds						
1,1-Dichloroethene	6.0×10^{-1}	0.71 mg/kg/day in drinking water (0.120 mg/kg/day)	Rat	Adrenal pheochromocytomas	C	IRIS 1993
1,1,1-Trichloroethane	NA				D	IRIS 1993
1,1,2-Trichloroethane	5.7×10^{-2}	139 mg/kg/day by gavage (9.3 mg/kg/day)	Mouse	Hepatocellular carcinomas	C	IRIS 1993
1,1,2,2-Tetrachloroethane	2.0×10^{-1}	87 mg/kg/day by gavage (6.56 mg/kg/day)	Mouse	Hepatocullular carcimonas	C	IRIS 1993
1,2-Dichloroethane	9.1×10^{-2}	47 mg/kg/day by gavage (4.46 mg/kg/day)	Rat	Hemangiosarcomas	B2	IRIS 1993
1,2-Dichloroethene	NA					IRIS 1993
1,2-Dichloropropane	6.8×10^{-2}	gavage	Mouse	Liver tumors	B2	HEAST 1992
2-Butanone (methyl ethyl ketone)	NA				D	IRIS 1993
Acetone	NA				D	IRIS 1993

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Benzene	2.9×10^{-2}	inhalation, occupational exposure	Human	Leukemia	A	IRIS 1993
Bromoform	7.9×10^{-3}	100 ppm (10.6 mg/kg/day) by gavage	Rat	Neoplastic lesions in the large intestines	B2	IRIS 1993
Carbon disulfide	NA					
Carbon tetrachloride	1.3×10^{-1}	0.95 mg/kg by gavage (1.02 mg/kg/day)	Hamsterm ouse rat	Hepatocellular carcinomas and hepatomas	B2	IRIS 1993
Chlorobenzene	NA				D	IRIS 1993
Chloroethane	NA					
Chloroform	6.1×10^{-3}	200 mg/L in drinking water (3.4 mg/kg/day)	Rat	Kidney tumors	B2	IRIS 1993
Chloromethane	1.3×10^{-2}	intermittent inhalation for 24 months	Mouse	Kidney tumors	C	HEAST 1992
Dibromochloromethane	8.4×10^{-2}	50 mg/kg/day by gavage (2.7 mg/kg/day)	Mouse	Hepatocellular adenomas, carcinomas	C	IRIS 1993
Ethylbenzene	NA				D	IRIS 1993

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Methylene chloride	7.5×10^{-3}	2,000 ppm by inhalation (122 mg/kg/day)	Mouse	Hepatocellular adenomas, carcinomas	B2	IRIS 1993
Styrene	NA					
Tetrachloroethene	5.2×10^{-2}	[not provided]				USEPA/ ECAO 1992
Toluene	NA				D	IRIS 1993
Trichloroethene	1.1×10^{-2}	[not provided]				USEPA 1992
Vinyl Chloride	1.9×10^{0}	In diet for 1,001 days	Rat	lung and liver tumors		HEAST 1992
Xylene (total)	NA				D	IRIS 1993
Semivolatile Compounds						
1,2-Dichlorobenzene	NA				D	IRIS 1993
1,2,4-Trichlorobenzene	NA				D	IRIS 1993
1,3-Dichlorobenzene	NA				D	IRIS 1993
1,4-Dichlorobenzene	2.4×10^{-2}	by gavage for 103 weeks	Mouse	Liver tumors	C	HEAST 1992

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
2-Chloronaphthalene	NA					
2-Chlorophenol	NA					
2-Methylnaphthalene	NA					
2-Methylphenol	NA					
2-Nitroaniline (o-)	NA					
2-Nitrophenol	NA					
2,4-Dichlorophenol	NA					
2,4-Dimethylphenol	NA					
2,4-Dinitrotoluene	6.8×10^{-1}	In diet	Rat	Liver; hepatocellular carcinomas; neoplastic nodules; mammary gland ademonas, fibroadenomas, fibromas, adenocarcinomas/ carcinoma	B2	IRIS 1993
2,4,6-Trichlorophenol	1.1×10^{-2}	5,000 ppm in diet (44.6 mg/kg/day)	Rat	Leukemia	B2	IRIS 1993

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
2,6-Dinitrotoluene	6.8×10^{-1}	In diet	Rat	Liver; hepatocellular carcinomas; neoplastic nodules; mammary gland ademonas, fibroadenomas, fibromas, adenocarcinomas/ carcinoma	B2	IRIS 1993
3-Nitroaniline (m-)	NA					
3,4-Dichloronitrobenzene	NA					
4-Chloro-3-methylphenol	NA					
4-Chloroaniline	NA					
4-Chlorophenyl-phenylether	NA					
4-Methylphenol	NA				C	IRIS 1993
4-Nitroaniline (p-)	NA					
Acenaphthene	NA					
Acenaphthylene	NA				D	IRIS 1993
Anthracene	NA				D	IRIS 1993
Benzoic acid	NA				D	IRIS 1993

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF			Weight of Evidence Classification	Source
		Exposure	Species	Effects of Concern		
Benzo(a)anthracene	7.3×10^{-1}	Estimated relative to BaP potency.			B2	USEPA, Region IV 1992
Benzo(a)pyrene	7.3×10^{0}	1 ppm in diet	Mouse	Squamous cell papillomas and carcinomas of the forestomach	B2	IRIS 1993
Benzo(b)fluoranthene	7.3×10^{-1}	Estimated relative to BaP potency.			B2	USEPA, Region IV 1992
Benzo(g,h,i)perylene	NA				D	IRIS 1993
Benzo(k)fluoranthene	7.3×10^{-1}	Estimated relative to BaP potency.			B2	USEPA, Region IV 1992
Benzyl alcohol	NA					
bis(2-Chloroethoxy)methane	NA				D	IRIS 1993
bis(2-Chloroethyl)ether	1.1×10^{0}	39 mg/kg/day by gavage followed by diet	Mouse	Hepatomas	B2	IRIS 1993
bis(2-Ethylhexyl)phathalate	1.4×10^{-2}	3,000 ppm in diet (32 mg/kg/day)	Mouse	Hepatocellular adenomas and carcinomas	B2	IRIS 1993

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Butylbenzylphthalate	NA				C	IRIS 1993
Carbazole	2.0×10^{-2}	oral for 96 wks	Mouse	Liver tumors	B2	HEAST 1992
Chrysene	7.3×10^{-2}	Estimated relative to BaP potency.			B2	USEPA, Region IV 1992
Dibenzofuran	NA				D	IRIS 1993
Diethylphthalate	NA				D	IRIS 1993
Dimethyl phthalate	NA				D	IRIS 1993
Di-n-butylphthalate	NA				D	IRIS 1993
Di-n-octylphthalate	NA					
Diphenylsulfone	NA					
Fluoranthene	NA				D	IRIS 1993
Fluorene	NA				D	IRIS 1993
Hexachlorobenzene	1.6×10^{0}	75 ppm in diet (0.73 mg/kg/day)	Rat	Hepatocellular carcinoma	B2	IRIS 1993
Hexachlorobutadiene	7.8×10^{-2}	20.0 mg/kg/day in diet (4.0 mg/kg/day)	Rat	Renal tubular adenomas and adenocarcinomas	C	IRIS 1993

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Hexachlorocyclopentadiene	NA				D	IRIS 1993
Hexachloroethane	1.4×10^{-2}	421 mg/kg/day by gavage 27.8 (mg/kg/day)	Mouse	Hepatocellular carcinomas	C	IRIS 1993
Indeno(1,2,3-cd)pyrene	7.3×10^{-1}	Estimated relative to BaP potency.			B2	USEPA, Region IV 1992
Naphthalene	NA				D	IRIS 1993
Nitrobenzene	NA				D	IRIS 1993
N-Nitrosodiphenylamine	4.9×10^{-3}	4,000 ppm in drinking water (30.6 mg/kg/day)	Rat	Transitional cell carcinoma of bladder	B2	IRIS 1993
N-Nitroso-di-n-propylamine	7.0×10^{0}	drinking water	Rat	Hepatocellular carcinoma	B2	IRIS 1993
Pentachlorophenol	1.2×10^{-1}	100 ppm in diet (1.4 mg/kg/day)	Mouse	Hepatocellular adenoma/carcinoma	B2	IRIS 1993
Phenanthrene	NA				D	IRIS 1993
Phenol	NA				D	IRIS 1993
Pyrene	NA				D	IRIS 1993

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Pesticides						
4,4'-DDD	2.4×10^{-1}	250 ppm in diet (245 mg/kg/day)	Mouse	Liver	B2	IRIS 1993
4,4'-DDE	3.4×10^{-1}	148 ppm in diet (0.90 mg/kg/day)	Mouse, hamster	Hepatocellular carcinomas	B2	IRIS 1993
4,4'-DDT	3.4×10^{-1}	diet	Mouse, rat	Liver	B2	IRIS 1993
Aldrin	1.7×10^{1}	10 ppm in diet (0.104 mg/kg/day)	Mouse	Liver carcinoma	B2	IRIS 1993
alpha-BHC	6.3×10^{0}	In diet for 24 weeks	Mouse	Liver tumors		IRIS 1993
beta-BHC	1.8×10^{0}	200 ppm in diet (1.96 mg/kg/day)	Mouse	Hepatic nodules and hepatocellular carcinomas	C	IRIS 1993
delta-BHC	NA				D	IRIS 1993
Chlordane (alpha-, gamma-)	1.3×10^{0}	25 ppm in diet (0.260 mg/kg/day)	Mouse	Hepatocellular carcinoma	B2	IRIS 1993
Dieldrin	1.6×10^{1}	diet	Mouse	Liver carcinoma	B2	IRIS 1993
Endosulfan (I and II)	NA					

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Endosulfan Sulfate	NA					
Endrin	NA				D	IRIS 1993
Endrin aldehyde	NA					
gamma-BHC (lindane)	1.3×10^{0}	In diet for 110 weeks	Mouse	Liver tumors	B2-C	HEAST 1992
Heptachlor	4.5×10^{0}	10 ppm in diet (0.108 mg/kg/day)	Mouse	Hepatocellular carcinomas	B2	IRIS 1993
Methoxychlor	NA				D	IRIS 1993
Other						
Kepone	NA				C	See Appendix F
Mirex	5.3×10^{-1}	diet		Hepatocellular adenomas or carcinomas	B2/C	See Appendix D
Photomirex	NA				D	See Appendix E

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Dioxins and Furans						
Total TCDD	NA	See TEF scheme at end of table.				USEPA 1989
2,3,7,8-TCDD	1.5×10^{5}	In diet for 720 days	Rat	Respiratory and lung tumors	B2	HEAST 1992
Total PeCDDs	NA	See TEF scheme at end of table.				USEPA 1989
Total HxCDDs	NA	See TEF scheme at end of table.				USEPA 1989
Total HpCDDs	NA	See TEF scheme at end of table.				USEPA 1989
1,2,3,4,6,7,8-HpCDD	NA	See TEF scheme at end of table.				USEPA 1989
OCDD	NA	See TEF scheme at end of table.				USEPA 1989
Total TCDFs	NA	See TEF scheme at end of table.				USEPA 1989
2,3,7,8-TCDF	NA	See TEF scheme at end of table.				USEPA 1989

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor (mg/kg/day)$^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Total PeCDF	NA	See TEF scheme at end of table.				USEPA 1989
1,2,3,7,8-PeCDF	NA	See TEF scheme at end of table.				USEPA 1989
2,3,4,7,8-PeCDF	NA	See TEF scheme at end of table.				USEPA 1989
Total HxCDF	NA	See TEF scheme at end of table.				USEPA 1989
1,2,3,4,7,8-HxCDF	NA	See TEF scheme at end of table.				USEPA 1989
1,2,3,6,7,8-HxCDF	NA	See TEF scheme at end of table.				USEPA 1989
2,3,4,6,7,8-HxCDF	NA	See TEF scheme at end of table.				USEPA 1989
Total HpCDF	NA	See TEF scheme at end of table.				USEPA 1989
1,2,3,4,6,7,8-HpCDF	NA	See TEF scheme at end of table.				USEPA 1989
1,2,3,4,7,8,9-HpCDF	NA	See TEF scheme at end of table.				USEPA 1989

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
OCDF	NA	See TEF scheme at end of table.				USEPA 1989
Inorganics						
Aluminum	NA					
Antimony	NA					
Arsenic	NA				A	IRIS 1993
Barium	NA					
Beryllium	4.3×10^{0}	5 ppm in drinking water	Rat	Gross TUmors all sties combined	B2	IRIS 1993
Cadmium	NA				B1	IRIS 1993
Calcium	ESS. NUTRIENT					
Chromium	NA				A	IRIS 1993
Cobalt	NA					
Copper	ESS. NUTRIENT					
Cyanide	NA				D	IRIS 1993
Iron	ESS. NUTRIENT					

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Lead	NA				B2	IRIS 1993
Magnesium	ESS. NUTRIENT					
Manganese	ESS. NUTRIENT					
Mercury	NA					
Nickel	NA					
Potassium	ESS. NUTRIENT					
Selenium	NA				D	IRIS 1993
Silver	NA				D	IRIS 1993
Sodium	ESS. NUTRIENT					
Thallium	NA					
Vanadium	NA					
Zinc	ESSENTIAL					

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source

NA Not Available

ESS. NUTRIENT: Chemical not evaluated in risk assessment because it is an essential nutrient.

HEAST: U.S. Environmental Protection Agency (USEPA). Office of Emergency and Remedial Response. Office of Research and Development. 1992. *Health effects assessment summary tables.* Annual: FY 1992; July 1992 and November 1992 Supplemental Updates. Washington, D.C.

IRIS: U.S. Environmental Protection Agency (USEPA). Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office. 1993. Integrated risk information system (IRIS). Cincinnati, OH.

TABLE A-2
Oral Slope Factors (SFs) for Chemicals Detected in Site Media

Compounds	Slope Factor $(mg/kg/day)^{-1}$	Basis for SF				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source

Toxicity Equivalence Factor (TEF) Approach for Chlorinated Dibenzo-p-dioxins and Dibenzofurans (CDDs and CDFs): The TEF procedure was used for CDDs and CDFs. This method relates the toxicity of CDDs and CDFs to 2,3,7,8-TCDD in the following fashion: CDD and CDF congener concentrations are multiplied by the TEFs listed below to express the concentration in terms of 2,3,7,8-TCDD equivalents. The products are summed to obtain the total 2,3,7,8-TCDD equivalents in the sample. The concentration, expressed in terms of 2,3,7,8-TCDD equivalents, is combined with exposure assumptions and the appropriate toxicity value for 2,3,7,8-TCDD to estimate the risk associated with the mixture of CDDs and CDFs.

CDD	TEF	CDF	TEF
		Mono-, Di-, TriCDFs	0
Mono-, Di-, or TriCDDs	0	2 3 7 8-TCDFs	0.1
2 3 7 8-TCDD	1	Other TCDFs	0
Other TCDDs	0	1 2 3 7 8-PeCDF	0.05
2 3 7 8-PeCDDs	0.5	2 3 4 7 8-PeCDFs	0.5
Other PeCDDs	0	Other PeCDFs	0
2 3 7 8-HxCDDs	0.1	2 3 7 8-HxCDFs	0.1
Other HxCDDs	0	Other HxCDFs	0
2 3 7 8-HpCDDs	0.01	2 3 7 8-HpCDFs	0.01
Other HpCDDs	0	Other HpCDFs	0
OCDD	0.001	OCDF	0.001

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TABLE A-3
Chronic Inhalation Reference Concentrations (RfCs)

Compounds	Chronic RfC (mg/m^3) [mg/kg/day]	Basis for RfC				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
1,1,1-Trichloroethane	1×10^{0} [3×10^{-1}]	500 ppm in diet for 6 months	Guinea Pig	Hepatotoxicity	1,000	HEAST 1992 (Table 2)
Bis(2-ethylhexyl)-phthalate	7×10^{-2} (converted from oral RfD[a]) [2×10^{-2}]	See Table A-1 for summary of RfD				
Carbon tetrachloride	2×10^{-3} (converted from oral RfD[a]) [7×10^{-4}]	See Table A-1 for summary of RfD				
Chlorobenzene	2×10^{-2} [6×10^{-3}]	Inhalation (intermittent) for 120 days	Rat	Liver and kidney effects	10,000	HEAST 1992 (Table 2)
Ethyl benzene	1×10^{0} [3×10^{-1}]	24-day inhalation exposure	Rabbit	Developmental toxicity	300	IRIS 1993
Styrene	1×10^{0} [3×10^{-1}]	8-hour inhalation exposure	Human	Cerebellar dysfunction	30	HEAST 1992
Trichloroethene	NA					
Xylene	7×10^{0} (converted from oral RfD[a]) [2×10^{0}]	See table A-1 for summary of RfD				
N-Nitrosodiphenylamine	NA					

TABLE A-3
Chronic Inhalation Reference Concentrations (RfCs)

Compounds	Chronic RfC (mg/m^3) [mg/kg/day]	Basis for RfC				
		Exposure	Species	Effects of Concern	Uncertainty Factor (Confidence Level)	Source
Mirex	7×10^{-4} (converted from oral RfD[a]) [2×10^{-4}]	See Table A-1 for summary of RfD				
Photomirex	5×10^{-3} (converted from oral RfD[a]) [1.3×10^{-3}]	See Table A-1 for summary of RfD				

[a] A USEPA-derived inhalation RfC was not available. Therefore, the RfC was approximated from the oral RfD using the assumptions of a 20 m^3/day breathing rate and 70 kg body weight.

HEAST: U.S. Environmental Protection Agency (USEPA). Office of Emergency and Remedial Response. Office of Research and Development. 1992. *Health effects assessment summary tables.* Annual: FY 1992; July 1992 and November 1992 Supplemental Updates. Washington, D.C.

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TABLE A-4
Inhalation Unit Risks

Compounds	Unit Risk $(\mu g/m^3)^{-1}$ [$(mg/kg/day)^{-1}$]	Basis for Unit Risk				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
1,1,1-Trichloroethane	NA				D	IRIS 1993
Bis(2-ethylhexyl)-phthalate	4×10^{-6} (converted from oral SF[a]) [1.4×10^{-2}]	See Table A-2 for summary of oral SF				
Carbon tetrachloride	1.5×10^{-5} [5.2×10^{-2}]	0.95 mg/kg by gavage (1.02 mg/kg/day)	Hamster, mouse, rat	Hepatocellular carcinomas and hepatomas	B2	IRIS 1993
Chlorobenzene	NA				D	IRIS 1993
Ethylbenzene	NA					
Styrene	NA					
Trichloroethene	1.7×10^{-6} [6.0×10^{-3}]					USEPA/ECAO 1992
Xylene	NA					
N-Nitrosodiphenylamine	1.4×10^{-6} (converted from oral SF[a]) [4.9×10^{-3}]	See Table A-2 for summary of oral SF			B2	IRIS 1993

TABLE A-4
Inhalation Unit Risks

Compounds	Unit Risk $(\mu g/m^3)^{-1}$ [$(mg/kg/day)^{-1}$]	Basis for Unit Risk				
		Exposure	Species	Effects of Concern	Weight of Evidence Classification	Source
Mirex	1.5×10^{-4} (converted from oral SF[a]) [5.3×10^{-1}]	See Table A-2 and Appendix D for a summary of the oral SF			B2/C	See Appendix D
Photomirex	NA					

[a] A USEPA-derived inhalation unit risk was not available. Therefore, the inhalation unit risk was approximated from the oral slope factor using the assumptions of a 20 m^3/day breathing rate and 70 kg body weight.

IRIS: U.S. Environmental Protection Agency (USEPA). Office of Health and Environmental Assessment. Environmental Criteria and Assessment Office. 1993. *Integrated risk information system (IRIS)*. Cincinnati, OH.

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APPENDIX B

Concentration and Toxicity Screen

APPENDIX B

Concentration and Toxicity Screen

This appendix presents details of the concentration and toxicity screen used to focus the assessment on the most significant chemicals. As stated in Chapter III of the EA, each chemical in a medium is scored according to its concentration and toxicity to obtain a risk factor as follows:

$$R_{ij} = (C_{ij}) (T_{ij})$$

where:

R_{ij} = risk factor for chemical i in medium j;

C_{ij} = maximum concentration of chemical i in medium j; and

T_{ij} = toxicity value for chemical i in medium j (either the slope factor or the reciprocal of the reference dose, 1/RfD).

Total chemical scores are calculated for each medium by summing all chemical-specific risk factors as follows:

$$R_j = R_{1j} + R_{2j} + R_{3j} + \ldots + R_{ij}$$

where:

R_j = total risk factor for medium j; and

$R_{1j} + \ldots + R_{ij}$ = risk factors for chemicals 1 through i in medium j.

A separate R_j is calculated for carcinogenic and noncarcinogenic effects for each medium. Chemicals whose R_{ij}/R_j ratios are very low compared with the ratios of other chemicals are eliminated from the risk assessment. As recommended in RAGS (p. 5-24), all chemicals in a medium whose R_{ij}/R_j ratios were less than 0.01 were eliminated from the risk assessment for that medium. The toxicity values used in calculating risk factors are presented in Appendix A. Of the 105 chemicals remaining in consideration for the quantitative risk assessment, a total of 19 chemicals could not be scored using the concentration and toxicity screen because no toxicity values were available. Thus, 86 chemicals were scored using the concentration and toxicity screen. The calculations conducted for the concentration and toxicity screen are summarized in Tables B-1 through B-13. A horizontal line drawn in each table indicated the cutoff between those chemicals with ratios above 0.01 and those chemicals with ratios below 0.01.

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TABLE B-1

SCORES FOR CHEMICALS DETECTED IN ON-SITE TEST PIT SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	2080000	2.0E-04	10,400,000,000
Hexachlorobutadiene	1400	2.0E-04	7,000,000
Hexachlorobenzene	2900	8.0E-04	3,625,000
1,2-Dichlorobenzene	290000	9.0E-02	3,222,222
Tetrachloroethene	13000	1.0E-02	1,300,000
Photomirex	559	1.3E-03	447,200
Hexachloroethane	400	1.0E-03	400,000
Trichloroethene	2200	6.0E-03	366,667
Dieldrin	13	5.0E-05	260,000
4,4'-DDT	100	5.0E-04	200,000
2,4-Dichlorophenol	430	3.0E-03	143,333
Kepone	85.8	6.5E-04	132,000
Arsenic	23.1	3.0E-04	77,000
Methoxychlor	380	5.0E-03	76,000
Endrin	16	3.0E-04	53,333
bis(2-Ethylhexyl)Phthalate	940	2.0E-02	47,000
1,1,2-Trichloroethane	180	4.0E-03	45,000
1,2-Dichloroethene (total)	350	9.0E-03	38,889
2-Chlorophenol	99	5.0E-03	19,800
4-Methylphenol	83	5.0E-03	16,600
1,4-Dichlorobenzene	3000	2.0E-01	15,000
2,3,7,8-TCDF	0.00015	1E-08	15,000
1,2,4-Trichlorobenzene	140	1.0E-02	14,000
Naphthalene	340	4.0E-02	8,500
Pyrene	240	3.0E-02	8,000
Vanadium	42.6	7.0E-03	6,086
Chlorobenzene	120	2.0E-02	6,000
Ethylbenzene	570	1.0E-01	5,700
Chromium	22.5	5.0E-03	4,500
Acetone	430	1.0E-01	4,300
Cadmium	1.6	5.0E-04	3,200
Fluoranthene	110	4.0E-02	2,750
Bromoform	35	2.0E-02	1,750
Nickel	34.9	2.0E-02	1,745
Barium	116	7.0E-02	1,657
Toluene	290	2.0E-01	1,450
Diethylphthalate	1100	8.0E-01	1,375
Chloroform	12	1.0E-02	1,200
Mercury	0.2	3.0E-04	667
OCDD	0.00037	1E-06	370
Phenol	220	6.0E-01	367
Selenium	1.3	5.0E-03	260
Beryllium	1.1	5.0E-03	220
Silver	0.7	5.0E-03	140
2-Butanone	78	6.0E-01	130
Benzoic Acid	490	4.0E+00	123

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Mirex	2080000	5.3E-01	1,102,400
Hexachlorobenzene	2900	1.6E+00	4,640
1,1,2,2-Tetrachloroethane	9000	2.0E-01	1,800
Benzo(a)Pyrene	190	7.3E+00	1,387
Tetrachloroethene	13000	5.2E-02	676
Benzo(k)Fluoranthene	390	7.3E-01	285
Benzo(b)Fluoranthene	340	7.3E-01	248
Dieldrin	13	1.6E+01	208
Hexachlorobutadiene	1400	7.8E-02	109
1,2-Dichloroethane	990	9.1E-02	90
1,4-Dichlorobenzene	3000	2.4E-02	72
Benzo(a)Anthracene	60	7.3E-01	44
Arsenic	23.1	1.8E+00	42
4,4'-DDT	100	3.4E-01	34
Benzene	900	2.9E-02	26
Trichloroethene	2200	1.1E-02	24
Chrysene	300	7.3E-02	22
bis(2-Ethylhexyl)Phthalate	940	1.4E-02	13
1,1,2-Trichloroethane	180	5.7E-02	10
Hexachloroethane	400	1.4E-02	6
1,2-Dichloropropane	73	6.8E-02	5
Beryllium	1.1	4.3E+00	5
2,3,7,8-TCDF	0.00015	1.5E+04	2
N-Nitrosodiphenylamine	440	4.9E-03	2
Bromoform	35	7.9E-03	0
Chloroform	12	6.1E-03	0
OCDD	0.00037	1.5E+02	0
1,2-Dichlorobenzene	290000	NA	NA
1,2-Dichloroethene (total)	350	NA	NA
1,2,4-Trichlorobenzene	140	NA	NA
2-Butanone	78	NA	NA
2-Chlorophenol	99	NA	NA
2,4-Dichlorophenol	430	NA	NA
4-Methylphenol	83	NA	NA
Acetone	430	NA	NA
Barium	116	NA	NA
Benzoic Acid	490	NA	NA
Cadmium	1.6	NA	NA
Chlorobenzene	120	NA	NA
Chromium	22.5	NA	NA
Cyanide	0.4	NA	NA
Diethylphthalate	1100	NA	NA
Endrin	16	NA	NA
Ethylbenzene	570	NA	NA
Fluoranthene	110	NA	NA
Kepone	85.8	NA	NA

TABLE B-1

SCORES FOR CHEMICALS DETECTED IN ON-SITE TEST PIT SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Cyanide	0.4	2.0E-02	20
Xylene (total)	13	2.0E+00	7
1,1,2,2-Tetrachloroethane	9000	NA	NA
1,2-Dichloroethane	990	NA	NA
1,2-Dichloropropane	73	NA	NA
Benzene	900	NA	NA
Benzo(a)Anthracene	60	NA	NA
Benzo(a)Pyrene	190	NA	NA
Benzo(b)Fluoranthene	340	NA	NA
Benzo(k)Fluoranthene	390	NA	NA
Chrysene	300	NA	NA
N-Nitrosodiphenylamine	440	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			10,417,574,560

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Mercury	0.2	NA	NA
Methoxychlor	380	NA	NA
Naphthalene	340	NA	NA
Nickel	34.9	NA	NA
Phenol	220	NA	NA
Photomirex	559	NA	NA
Pyrene	240	NA	NA
Selenium	1.3	NA	NA
Silver	0.7	NA	NA
Toluene	290	NA	NA
Vanadium	42.6	NA	NA
Xylene (total)	13	NA	NA
TOTAL CARCINOGENIC FACTOR =			1,112,150

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-2

SCORES FOR CHEMICALS DETECTED IN ON-SITE POND SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	938000	2.0E-04	4,690,000,000
Tetrachloroethene	38000000	1.0E-02	3,800,000,000
Trichloroethene	2200000	6.0E-03	366,666,667
Hexachloroethane	330000	1.0E-03	330,000,000
Hexachlorobutadiene	56000	2.0E-04	280,000,000
Hexachlorobenzene	100000	8.0E-04	125,000,000
Carbon Tetrachloride	70000	7.0E-04	100,000,000
Antimony	36700	4.0E-04	91,750,000
Dieldrin	3000	5.0E-05	60,000,000
Arsenic	17800	3.0E-04	59,333,333
Methoxychlor	280000	5.0E-03	56,000,000
1,2-Dichlorobenzene	2700000	9.0E-02	30,000,000
Chlorobenzene	300000	2.0E-02	15,000,000
Chromium	47500	5.0E-03	9,500,000
2,3,7,8-TCDF	0.071	1E-08	7,100,000
Chloroform	45000	1.0E-02	4,500,000
Vanadium	31400	7.0E-03	4,485,714
OCDD	3	1E-06	3,000,000
Toluene	550000	2.0E-01	2,750,000
Styrene	420000	2.0E-01	2,100,000
Cadmium	890	5.0E-04	1,780,000
Bromoform	34000	2.0E-02	1,700,000
Barium	115000	7.0E-02	1,642,857
Nickel	29800	2.0E-02	1,490,000
2,4-Dichlorophenol	3900	3.0E-03	1,300,000
Kepone	761	6.5E-04	1,170,769
Mercury	300	3.0E-04	1,000,000
Acetone	98000	1.0E-01	980,000
Ethylbenzene	57000	1.0E-01	570,000
Dibromochloromethane	8700	2.0E-02	435,000
Selenium	2100	5.0E-03	420,000
1,2,4-Trichlorobenzene	3700	1.0E-02	370,000
bis(2-Ethylhexyl)Phthalate	4000	2.0E-02	200,000
4,4'-DDT	96	5.0E-04	192,000
Beryllium	940	5.0E-03	188,000
Benzoic Acid	570000	4.0E+00	142,500
1,4-Dichlorobenzene	27000	2.0E-01	135,000
Xylene (total)	250000	2.0E+00	125,000
Naphthalene	3200	4.0E-02	80,000
Cyanide	1400	2.0E-02	70,000
Photomirex	33.4	1.3E-03	26,720
Diethylphthalate	19000	8.0E-01	23,750
1,2-Dichloroethene (total)	10	9.0E-03	1,111
Phenol	270	6.0E-01	450
1,1,2,2-Tetrachloroethane	7200000	NA	NA
1,2-Dichloroethane	250000	NA	NA

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Tetrachloroethene	38000000	5.2E-02	1,976,000
1,1,2,2-Tetrachloroethane	7200000	2.0E-01	1,440,000
Mirex	938000	5.3E-01	497,140
Hexachlorobenzene	100000	1.6E+00	160,000
Benzene	4700000	2.9E-02	136,300
Dieldrin	3000	1.6E+01	48,000
Arsenic	17800	1.8E+00	32,040
Trichloroethene	2200000	1.1E-02	24,200
1,2-Dichloroethane	250000	9.1E-02	22,750
Carbon Tetrachloride	70000	1.3E-01	9,100
Hexachloroethane	330000	1.4E-02	4,620
Hexachlorobutadiene	56000	7.8E-02	4,368
Beryllium	940	4.3E+00	4,042
2,3,7,8-TCDF	0.071	1.5E+04	1,065
Dibromochloromethane	8700	8.4E-02	731
1,4-Dichlorobenzene	27000	2.4E-02	648
OCDD	3	1.5E+02	450
Chloroform	45000	6.1E-03	275
Bromoform	34000	7.9E-03	269
N-Nitrosodiphenylamine	13000	4.9E-03	64
bis(2-Ethylhexyl)Phthalate	4000	1.4E-02	56
4,4'-DDT	96	3.4E-01	33
Chloromethane	1500	1.3E-02	20
1,2-Dichloropropane	72	6.8E-02	5
1,2-Dichlorobenzene	2700000	NA	NA
1,2-Dichloroethene (total)	10	NA	NA
1,2,4-Trichlorobenzene	3700	NA	NA
2,4-Dichlorophenol	3900	NA	NA
Acetone	98000	NA	NA
Antimony	36700	NA	NA
Barium	115000	NA	NA
Benzoic Acid	570000	NA	NA
Cadmium	890	NA	NA
Chlorobenzene	300000	NA	NA
Chromium	47500	NA	NA
Cyanide	1400	NA	NA
Diethylphthalate	19000	NA	NA
Ethylbenzene	57000	NA	NA
Kepone	761	NA	NA
Mercury	300	NA	NA
Methoxychlor	280000	NA	NA
Naphthalene	3200	NA	NA
Nickel	29800	NA	NA
Phenol	270	NA	NA
Photomirex	33.4	NA	NA
Selenium	2100	NA	NA

TABLE B-2

SCORES FOR CHEMICALS DETECTED IN ON-SITE POND SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
1,2-Dichloropropane	72	NA	NA
Benzene	4700000	NA	NA
Chloromethane	1500	NA	NA
N-Nitrosodiphenylamine	13000	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			10,051,228,872

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Styrene	420000	NA	NA
Toluene	550000	NA	NA
Vanadium	31400	NA	NA
Xylene (total)	250000	NA	NA
TOTAL CARCINOGENIC FACTOR =			4,362,174

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-3

SCORES FOR CHEMICALS DETECTED IN GROUND WATER SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Tetrachloroethene	100000	1.0E-02	10,000,000
Trichloroethene	20000	6.0E-03	3,333,333
1,2-Dichloroethene (total)	19000	9.0E-03	2,111,111
Mirex	239.6	2.0E-04	1,198,000
2-Nitroaniline	68	6.0E-05	1,133,333
Hexachlorobutadiene	110	2.0E-04	550,000
Hexachloroethane	470	1.0E-03	470,000
1,2-Dichlorobenzene	36000	9.0E-02	400,000
Cadmium	123	5.0E-04	246,000
Chlorobenzene	4700	2.0E-02	235,000
Arsenic	70.4	3.0E-04	234,667
2,4-Dichlorophenol	670	3.0E-03	223,333
Hexachlorobenzene	130	8.0E-04	162,500
2,3,4,7,8-PeCDF	0.000272	2E-09	136,000
Chloroform	1200	1.0E-02	120,000
Toluene	23000	2.0E-01	115,000
Nickel	2265.1	2.0E-02	113,255
Vanadium	635.85	7.0E-03	90,836
bis(2-Ethylhexyl)Phthalate	920	2.0E-02	46,000
Chromium	171.2	5.0E-03	34,240
Bromoform	680	2.0E-02	34,000
1,2,3,4,7,8-HxCDF	0.000283	1E-08	28,300
1,1,2-Trichloroethane	110	4.0E-03	27,500
2,3,7,8-TCDF	0.000231	1E-08	23,100
Kepone	13.1	6.5E-04	20,154
2,3,7,8-TCDD	0.0000167	1E-09	16,700
1,2,3,6,7,8-HxCDF	0.00016	1E-08	16,000
Beryllium	78	5.0E-03	15,600
Antimony	5.9	4.0E-04	14,750
Endrin	4.4	3.0E-04	14,667
1,1-Dichloroethene	130	9.0E-03	14,444
Ethylbenzene	1200	1.0E-01	12,000
2,3,4,6,7,8-HxCDF	0.000119	1E-08	11,900
4-Methylphenol	42	5.0E-03	8,400
gamma-Chlordane	0.44	6.0E-05	7,333
4,4'-DDT	3.2	5.0E-04	6,400
Mercury	1.77	3.0E-04	5,900
Acetone	530	1.0E-01	5,300
1,2,3,4,6,7,8-HpCDF	0.000526	1E-07	5,260
Silver	24.69	5.0E-03	4,938
1,2,3,7,8-PeCDF	0.000098	2E-08	4,900
Photomirex	4.83	1.3E-03	3,864
2-Chlorophenol	18	5.0E-03	3,600
Barium	239.83	7.0E-02	3,426
Naphthalene	97	4.0E-02	2,425
1,2,3,4,6,7,8-HpCDD	0.000181	1E-07	1,810

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
1,1,2,2-Tetrachloroethane	60000	2.0E-01	12,000
Tetrachloroethene	100000	5.2E-02	5,200
Vinyl Chloride	1700	1.9E+00	3,230
1,2-Dichloroethane	23000	9.1E-02	2,093
Benzene	45000	2.9E-02	1,305
Beryllium	78	4.3E+00	335
Trichloroethene	20000	1.1E-02	220
Hexachlorobenzene	130	1.6E+00	208
Mirex	239.6	5.3E-01	127
Arsenic	70.4	1.8E+00	127
1,1-Dichloroethene	130	6.0E-01	78
2,3,4,7,8-PeCDF	0.000272	7.5E+04	20
bis(2-Ethylhexyl)Phthalate	920	1.4E-02	13
4,4'-DDD	53	2.4E-01	13
Hexachlorobutadiene	110	7.8E-02	9
Chloroform	1200	6.1E-03	7
1,4-Dichlorobenzene	300	2.4E-02	7
Hexachloroethane	470	1.4E-02	7
1,1,2-Trichloroethane	110	5.7E-02	6
Bromoform	680	7.9E-03	5
1,2,3,4,7,8-HxCDF	0.000283	1.5E+04	4
2,3,7,8-TCDF	0.000231	1.5E+04	3
2,3,7,8-TCDD	0.0000167	1.5E+05	3
1,2,3,6,7,8-HxCDF	0.00016	1.5E+04	2
2,3,4,6,7,8-HxCDF	0.000119	1.5E+04	2
4,4'-DDT	3.2	3.4E-01	1
1,2,3,4,6,7,8-HpCDF	0.000526	1.5E+03	1
1,2,3,7,8-PeCDF	0.000098	7.5E+03	1
Chloromethane	47	1.3E-02	1
gamma-Chlordane	0.44	1.3E+00	1
Dieldrin	0.018	1.6E+01	0
1,2,3,4,6,7,8-HpCDD	0.000181	1.5E+03	0
1,2,3,4,7,8,9-HpCDF	0.000112	1.5E+03	0
Heptachlor	0.024	4.5E+00	0
OCDD	0.000532	1.5E+02	0
OCDF	0.000486	1.5E+02	0
2,4,6-Trichlorophenol	6	1.1E-02	0
1,2-Dichlorobenzene	36000	NA	NA
1,2-Dichloroethene (total)	19000	NA	NA
1,2,4-Trichlorobenzene	18	NA	NA
2-Chlorophenol	18	NA	NA
2-Nitroaniline	68	NA	NA
2,4-Dichlorophenol	670	NA	NA
4-Methylphenol	42	NA	NA
Acenaphthene	6	NA	NA
Acetone	530	NA	NA

TABLE B-3

SCORES FOR CHEMICALS DETECTED IN GROUND WATER SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
1,2,4-Trichlorobenzene	18	1.0E-02	1,800
1,4-Dichlorobenzene	300	2.0E-01	1,500
Cyanide	25.3	2.0E-02	1,265
1,2,3,4,7,8,9-HpCDF	0.000112	1E-07	1,120
Phenol	400	6.0E-01	667
OCDD	0.000532	1E-06	532
OCDF	0.000486	1E-06	486
Methoxychlor	2.4	5.0E-03	480
Dieldrin	0.018	5.0E-05	360
Acenaphthene	6	6.0E-02	100
Heptachlor	0.024	5.0E-04	48
Pyrene	1	3.0E-02	33
Styrene	6	2.0E-01	30
Diethylphthalate	1	8.0E-01	1
1,1,2,2-Tetrachloroethane	60000	NA	NA
1,2-Dichloroethane	23000	NA	NA
2,4,6-Trichlorophenol	6	NA	NA
4,4'-DDD	53	NA	NA
Benzene	45000	NA	NA
Chloromethane	47	NA	NA
Vinyl Chloride	1700	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			21,273,702

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Antimony	5.9	NA	NA
Barium	239.83	NA	NA
Cadmium	123	NA	NA
Chlorobenzene	4700	NA	NA
Chromium	171.2	NA	NA
Cyanide	25.3	NA	NA
Diethylphthalate	1	NA	NA
Endrin	4.4	NA	NA
Ethylbenzene	1200	NA	NA
Kepone	13.1	NA	NA
Mercury	1.77	NA	NA
Methoxychlor	2.4	NA	NA
Naphthalene	97	NA	NA
Nickel	2265.1	NA	NA
Phenol	400	NA	NA
Photomirex	4.83	NA	NA
Pyrene	1	NA	NA
Silver	24.69	NA	NA
Styrene	6	NA	NA
Toluene	23000	NA	NA
Vanadium	635.85	NA	NA
TOTAL CARCINOGENIC FACTOR =			25,030

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-4

SCORES FOR CHEMICALS DETECTED IN AIR SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (mg/m^3)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
bis(2-Ethylhexyl)Phthalate	2.55E-02	2.0E-02	1.3E+00
Mirex	4.70E-05	2.0E-04	2.3E-01
Carbon Tetrachloride	1.67E-05	7.0E-04	2.4E-02
Photomirex	5.63E-06	1.3E-03	4.5E-03
Chlorobenzene	1.56E-05	6.0E-03	2.6E-03
1,1,1-Trichloroethane	7.44E-05	3.0E-01	2.5E-04
Ethylbenzene	1.43E-05	3.0E-01	4.8E-05
Styrene	8.80E-06	3.0E-01	2.9E-05
Xylene (total)	5.71E-05	2.0E+00	2.9E-05
N-Nitrosodiphenylamine	2.20E-03	NA	NA
Trichloroethene	1.52E-05	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			1.5E+00

CHEMICAL	MAXIMUM CONCENTRATION (mg/m^3)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Bis-(2-Ethylhexyl)Phthalate	2.55E-02	1.4E-02	3.6E-04
Mirex	4.70E-05	5.3E-01	2.5E-05
N-Nitrosodiphenylamine	2.20E-03	4.9E-03	1.1E-05
Carbon Tetrachloride	1.67E-05	5.3E-02	8.9E-07
Trichloroethene	1.52E-05	6.0E-03	9.1E-08
1,1,1-Trichloroethane	7.44E-05	NA	NA
Chlorobenzene	1.56E-05	NA	NA
Ethylbenzene	1.43E-05	NA	NA
Photomirex	5.63E-06	NA	NA
Styrene	8.80E-06	NA	NA
Xylene (total)	5.71E-05	NA	NA
TOTAL CARCINOGENIC FACTOR =			3.9E-04

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-5

SCORES FOR CHEMICALS DETECTED IN ON-SITE SEDIMENT SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	129000	2.0E-04	645,000,000
Hexachlorobenzene	3000	8.0E-04	3,750,000
Hexachlorobutadiene	230	2.0E-04	1,150,000
Photomirex	530	1.3E-03	424,000
Methoxychlor	1600	5.0E-03	320,000
Dieldrin	13	5.0E-05	260,000
Hexachloroethane	220	1.0E-03	220,000
Tetrachloroethene	1900	1.0E-02	190,000
2,4-Dichlorophenol	420	3.0E-03	140,000
1,2-Dichlorobenzene	6800	9.0E-02	75,556
Trichloroethene	420	6.0E-03	70,000
1,2-Dichloroethene (total)	280	9.0E-03	31,111
Pyrene	920	3.0E-02	30,667
Fluoranthene	730	4.0E-02	18,250
Naphthalene	260	4.0E-02	6,500
1,1,2-Trichloroethane	10	4.0E-03	2,500
Diethylphthalate	1300	8.0E-01	1,625
Chloroform	16	1.0E-02	1,600
1,4-Dichlorobenzene	260	2.0E-01	1,300
Fluorene	45	4.0E-02	1,125
Chlorobenzene	20	2.0E-02	1,000
Acenaphthene	33	6.0E-02	550
Anthracene	110	3.0E-01	367
Phenol	60	6.0E-01	100
1,1,1-Trichloroethane	5	9.0E-02	56
Toluene	8	2.0E-01	40
2-Butanone	4	6.0E-01	7
Xylene (total)	4	2.0E+00	2
1,1,2,2-Tetrachloroethane	370	NA	NA
1,2-Dichloroethane	640	NA	NA
1,2-Dichloropropane	3	NA	NA
2,4,6-Trichlorophenol	95	NA	NA
4,4'-DDD	11	NA	NA
Benzene	59	NA	NA
Benzo(a)Anthracene	410	NA	NA
Benzo(a)Pyrene	310	NA	NA
Benzo(b)Fluoranthene	470	NA	NA
Benzo(k)Fluoranthene	370	NA	NA
Chrysene	510	NA	NA
Indeno(1,2,3-cd)Pyrene	24	NA	NA
Vinyl Chloride	14	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			651,696,354

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Mirex	129000	5.3E-01	68,370
Hexachlorobenzene	3000	1.6E+00	4,800
Benzo(a)Pyrene	310	7.3E+00	2,263
Benzo(b)Fluoranthene	470	7.3E-01	343
Benzo(a)Anthracene	410	7.3E-01	299
Benzo(k)Fluoranthene	370	7.3E-01	270
Dieldrin	13	1.6E+01	208
Tetrachloroethene	1900	5.2E-02	99
1,1,2,2-Tetrachloroethane	370	2.0E-01	74
1,2-Dichloroethane	640	9.1E-02	58
Chrysene	510	7.3E-02	37
Vinyl Chloride	14	1.9E+00	27
Hexachlorobutadiene	230	7.8E-02	18
Indeno(1,2,3-cd)Pyrene	24	7.3E-01	18
Trichloroethene	420	1.1E-02	5
Hexachloroethane	220	1.4E-02	3
4,4'-DDD	11	2.4E-01	3
Benzene	59	2.9E-02	2
2,4,6-Trichlorophenol	95	1.1E-02	1
1,1,2-Trichloroethane	10	5.7E-02	1
1,2-Dichloropropane	3	6.8E-02	0
Chloroform	16	6.1E-03	0
1,1,1-Trichloroethane	5	NA	NA
1,2-Dichlorobenzene	6800	NA	NA
1,2-Dichloroethene (total)	280	NA	NA
1,4-Dichlorobenzene	260	NA	NA
2-Butanone	4	NA	NA
2,4-Dichlorophenol	420	NA	NA
Acenaphthene	33	NA	NA
Anthracene	110	NA	NA
Chlorobenzene	20	NA	NA
Diethylphthalate	1300	NA	NA
Fluoranthene	730	NA	NA
Fluorene	45	NA	NA
Methoxychlor	1600	NA	NA
Naphthalene	260	NA	NA
Phenol	60	NA	NA
Photomirex	530	NA	NA
Pyrene	920	NA	NA
Toluene	8	NA	NA
Xylene (total)	4	NA	NA
TOTAL CARCINOGENIC FACTOR =			76,898

TABLE B-5

SCORES FOR CHEMICALS DETECTED IN ON-SITE SEDIMENT SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR	CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-6

SCORES FOR CHEMICALS DETECTED IN ON-SITE SURFACE WATER SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Tetrachloroethene	340	1.0E-02	34,000
Trichloroethene	94	6.0E-03	15,667
Hexachloroethane	5	1.0E-03	5,000
Carbon Tetrachloride	2	7.0E-04	2,857
1,2-Dichlorobenzene	180	9.0E-02	2,000
Mirex	0.362	2.0E-04	1,810
Acetone	170	1.0E-01	1,700
1,2-Dichloroethene (total)	14	9.0E-03	1,556
2,4-Dichlorophenol	4	3.0E-03	1,333
1,1,2-Trichloroethane	5	4.0E-03	1,250
Chlorobenzene	13	2.0E-02	650
Kepone	0.292	6.5E-04	449
Bromoform	6	2.0E-02	300
Chloroform	3	1.0E-02	300
Methoxychlor	0.67	5.0E-03	134
Toluene	25	2.0E-01	125
Dibromochloromethane	2	2.0E-02	100
Benzoic Acid	310	4.0E+00	78
Benzyl Alcohol	11	3.0E-01	37
bis(2-Ethylhexyl)Phthalate	0.6	2.0E-02	30
Ethylbenzene	2	1.0E-01	20
Photomirex	0.0151	1.3E-03	12
Xylene (total)	6	2.0E+00	3
1,1,2,2-Tetrachloroethane	470	NA	NA
1,2-Dichloroethane	100	NA	NA
Benzene	140	NA	NA
Chloromethane	3	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			69,410

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
1,1,2,2-Tetrachloroethane	470	2.0E-01	94
Tetrachloroethene	340	5.2E-02	18
1,2-Dichloroethane	100	9.1E-02	9
Benzene	140	2.9E-02	4
Trichloroethene	94	1.1E-02	1
1,1,2-Trichloroethane	5	5.7E-02	0
Carbon Tetrachloride	2	1.3E-01	0
Mirex	0.362	5.3E-01	0
Dibromochloromethane	2	8.4E-02	0
Hexachloroethane	5	1.4E-02	0
Bromoform	6	7.9E-03	0
Chloromethane	3	1.3E-02	0
Chloroform	3	6.1E-03	0
bis(2-Ethylhexyl)Phthalate	0.6	1.4E-02	0
1,2-Dichlorobenzene	180	NA	NA
1,2-Dichloroethene (total)	14	NA	NA
2,4-Dichlorophenol	4	NA	NA
Acetone	170	NA	NA
Benzoic Acid	310	NA	NA
Benzyl Alcohol	11	NA	NA
Chlorobenzene	13	NA	NA
Ethylbenzene	2	NA	NA
Kepone	0.292	NA	NA
Methoxychlor	0.67	NA	NA
Photomirex	0.0151	NA	NA
Toluene	25	NA	NA
Xylene (total)	6	NA	NA
TOTAL CARCINOGENIC FACTOR =			127

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-7

SCORES FOR CHEMICALS DETECTED IN OFF-SITE SOIL SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	891	2.0E-04	4,455,000
Dieldrin	60	5.0E-05	1,200,000
4,4'-DDT	110	5.0E-04	220,000
bis(2-Ethylhexyl)Phthalate	4100	2.0E-02	205,000
Pyrene	2000	3.0E-02	66,667
Arsenic	18.7	3.0E-04	62,333
4-Methylphenol	270	5.0E-03	54,000
Fluoranthene	2100	4.0E-02	52,500
Antimony	13.1	4.0E-04	32,750
Chromium	128	5.0E-03	25,600
Photomirex	15.4	1.3E-03	12,320
Naphthalene	270	4.0E-02	6,750
Cadmium	3.3	5.0E-04	6,600
Mercury	1.8	3.0E-04	6,000
Barium	280	7.0E-02	4,000
Vanadium	24.5	7.0E-03	3,500
Nickel	60.8	2.0E-02	3,040
Silver	10.2	5.0E-03	2,040
Fluorene	64	4.0E-02	1,600
Anthracene	400	3.0E-01	1,333
Acenaphthene	49	6.0E-02	817
1,4-Dichlorobenzene	88	2.0E-01	440
Cyanide	3.7	2.0E-02	185
Beryllium	0.91	5.0E-03	182
Benzoic Acid	450	4.0E+00	113
Selenium	0.52	5.0E-03	104
Diethylphthalate	70	8.0E-01	88
Xylene (total)	6	2.0E+00	3
1,2-Dichloroethane	6	NA	NA
1,2-Dichloropropane	90	NA	NA
4,4'-DDD	111	NA	NA
Benzo(a)Anthracene	1400	NA	NA
Benzo(a)Pyrene	1200	NA	NA
Benzo(b)Fluoranthene	2400	NA	NA
Benzo(k)Fluoranthene	2400	NA	NA
Chrysene	1300	NA	NA
Indeno(1,2,3-cd)Pyrene	420	NA	NA
N-Nitrosodiphenylamine	200	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			6,422,964

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Benzo(a)Pyrene	1200	7.3E+00	8,760
Benzo(b)Fluoranthene	2400	7.3E-01	1,752
Benzo(k)Fluoranthene	2400	7.3E-01	1,752
Benzo(a)Anthracene	1400	7.3E-01	1,022
Dieldrin	60	1.6E+01	960
Mirex	891	5.3E-01	472
Indeno(1,2,3-cd)Pyrene	420	7.3E-01	307
Chrysene	1300	7.3E-02	95
bis(2-Ethylhexyl)Phthalate	4100	1.4E-02	57
4,4'-DDT	110	3.4E-01	37
Arsenic	18.7	1.8E+00	34
4,4'-DDD	111	2.4E-01	27
1,2-Dichloropropane	90	6.8E-02	6
Beryllium	0.91	4.3E+00	4
1,4-Dichlorobenzene	88	2.4E-02	2
N-Nitrosodiphenylamine	200	4.9E-03	1
1,2-Dichloroethane	6	9.1E-02	1
4-Methylphenol	270	NA	NA
Acenaphthene	49	NA	NA
Anthracene	400	NA	NA
Antimony	13.1	NA	NA
Barium	280	NA	NA
Benzoic Acid	450	NA	NA
Cadmium	3.3	NA	NA
Chromium	128	NA	NA
Cyanide	3.7	NA	NA
Diethylphthalate	70	NA	NA
Fluoranthene	2100	NA	NA
Fluorene	64	NA	NA
Mercury	1.8	NA	NA
Naphthalene	270	NA	NA
Nickel	60.8	NA	NA
Photomirex	15.4	NA	NA
Pyrene	2000	NA	NA
Selenium	0.52	NA	NA
Silver	10.2	NA	NA
Vanadium	24.5	NA	NA
Xylene (total)	6	NA	NA
TOTAL CARCINOGENIC FACTOR =			15,289

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

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TABLE B-8

SCORES FOR CHEMICALS DETECTED IN CRANE-DEMING SOIL SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	100	2.0E-04	500,000
Photomirex	5.65	1.3E-03	4,520
Pyrene	66	3.0E-02	2,200
Fluoranthene	47	4.0E-02	1,175
Naphthalene	28	4.0E-02	700
Tetrachloroethene	2	1.0E-02	200
Toluene	3	2.0E-01	15
Benzo(a)Pyrene	34	NA	NA
Benzo(b)Fluoranthene	36	NA	NA
Benzo(k)Fluoranthene	30	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			508,810

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Benzo(a)Pyrene	34	7.3E+00	248
Mirex	100	5.3E-01	53
Benzo(b)Fluoranthene	36	7.3E-01	26
Benzo(k)Fluoranthene	30	7.3E-01	22
Tetrachloroethene	2	5.2E-02	0
Fluoranthene	47	NA	NA
Naphthalene	28	NA	NA
Photomirex	5.65	NA	NA
Pyrene	66	NA	NA
Toluene	3	NA	NA
TOTAL CARCINOGENIC FACTOR =			349

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-9

SCORES FOR CHEMICALS DETECTED IN RR TEST PIT SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	2230	2.0E-04	11,150,000
1,2-Dichlorobenzene	34000	9.0E-02	377,778
Kepone	65.3	6.5E-04	100,462
2,4-Dichlorophenol	190	3.0E-03	63,333
Photomirex	44.8	1.3E-03	35,840
1,2,4-Trichlorobenzene	290	1.0E-02	29,000
1,1,2-Trichloroethane	110	4.0E-03	27,500
Naphthalene	850	4.0E-02	21,250
Trichloroethene	110	6.0E-03	18,333
Pyrene	360	3.0E-02	12,000
Fluoranthene	420	4.0E-02	10,500
Tetrachloroethene	98	1.0E-02	9,800
bis(2-Ethylhexyl)Phthalate	160	2.0E-02	8,000
1,4-Dichlorobenzene	700	2.0E-01	3,500
1,2-Dichloroethene (total)	12	9.0E-03	1,333
Chlorobenzene	25	2.0E-02	1,250
Toluene	60	2.0E-01	300
Chloroform	2	1.0E-02	200
Anthracene	51	3.0E-01	170
Diethylphthalate	57	8.0E-01	71
1,1,2,2-Tetrachloroethane	710	NA	NA
Benzene	510	NA	NA
Benzo(a)Anthracene	180	NA	NA
Benzo(a)Pyrene	180	NA	NA
Benzo(b)Fluoranthene	540	NA	NA
Benzo(k)Fluoranthene	540	NA	NA
Chrysene	330	NA	NA
Indeno(1,2,3-cd)Pyrene	200	NA	NA
N-Nitrosodiphenylamine	750	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			11,870,621

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Benzo(a)Pyrene	180	7.3E+00	1,314
Mirex	2230	5.3E-01	1,182
Benzo(b)Fluoranthene	540	7.3E-01	394
Benzo(k)Fluoranthene	540	7.3E-01	394
Indeno(1,2,3-cd)Pyrene	200	7.3E-01	146
1,1,2,2-Tetrachloroethane	710	2.0E-01	142
Benzo(a)Anthracene	180	7.3E-01	131
Chrysene	330	7.3E-02	24
Benzene	510	2.9E-02	15
1,1,2-Trichloroethane	110	5.7E-02	6
Tetrachloroethene	98	5.2E-02	5
N-Nitrosodiphenylamine	750	4.9E-03	4
bis(2-Ethylhexyl)Phthalate	160	1.4E-02	2
Trichloroethene	110	1.1E-02	1
Chloroform	2	6.1E-03	0
1,2-Dichlorobenzene	34000	NA	NA
1,2-Dichloroethene (total)	12	NA	NA
1,2,4-Trichlorobenzene	290	NA	NA
1,4-Dichlorobenzene	700	NA	NA
2,4-Dichlorophenol	190	NA	NA
Anthracene	51	NA	NA
Chlorobenzene	25	NA	NA
Diethylphthalate	57	NA	NA
Fluoranthene	420	NA	NA
Kepone	65.3	NA	NA
Naphthalene	850	NA	NA
Photomirex	44.8	NA	NA
Pyrene	360	NA	NA
Toluene	60	NA	NA
TOTAL CARCINOGENIC FACTOR =			3,761

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-10

SCORES FOR CHEMICALS DETECTED IN MFLBC SURFACE WATER SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
bis(2-Ethylhexyl)Phthalate	6	2.0E-02	300
Chloromethane	3	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			300

CHEMICAL	MAXIMUM CONCENTRATION (ug/l)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
bis(2-Ethylhexyl)Phthalate	6	1.4E-02	0.084
Chloromethane	3	1.3E-02	0.039
TOTAL CARCINOGENIC FACTOR =			0.123

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-11

SCORES FOR CHEMICALS DETECTED IN MFLBC SEDIMENT SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	2820	2.0E-04	14,100,000
4-Methylphenol	2800	5.0E-03	560,000
4,4'-DDT	250	5.0E-04	500,000
bis(2-Ethylhexyl)Phthalate	1800	2.0E-02	90,000
Fluoranthene	1100	4.0E-02	27,500
Pyrene	790	3.0E-02	26,333
Heptachlor	9.4	5.0E-04	18,800
Photomirex	7.38	1.3E-03	5,904
Fluorene	230	4.0E-02	5,750
Naphthalene	140	4.0E-02	3,500
Acenaphthene	100	6.0E-02	1,667
Anthracene	340	3.0E-01	1,133
Acetone	80	1.0E-01	800
Phenol	160	6.0E-01	267
Benzoic Acid	430	4.0E+00	108
2-Butanone	10	6.0E-01	17
1,2-Dichloroethane	2	NA	NA
1,2-Dichloropropane	18	NA	NA
Benzo(a)Anthracene	480	NA	NA
Benzo(a)Pyrene	310	NA	NA
Benzo(b)Fluoranthene	680	NA	NA
Benzo(k)Fluoranthene	680	NA	NA
Chrysene	530	NA	NA
Indeno(1,2,3-cd)Pyrene	150	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			15,341,778

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Benzo(a)Pyrene	310	7.3E+00	2,263
Mirex	2820	5.3E-01	1,495
Benzo(b)Fluoranthene	680	7.3E-01	496
Benzo(k)Fluoranthene	680	7.3E-01	496
Benzo(a)Anthracene	480	7.3E-01	350
Indeno(1,2,3-cd)Pyrene	150	7.3E-01	110
4,4'-DDT	250	3.4E-01	85
Heptachlor	9.4	4.5E+00	42
Chrysene	530	7.3E-02	39
bis(2-Ethylhexyl)Phthalate	1800	1.4E-02	25
1,2-Dichloropropane	18	6.8E-02	1
1,2-Dichloroethane	2	9.1E-02	0
2-Butanone	10	NA	NA
4-Methylphenol	2800	NA	NA
Acenaphthene	100	NA	NA
Acetone	80	NA	NA
Anthracene	340	NA	NA
Benzoic Acid	430	NA	NA
Fluoranthene	1100	NA	NA
Fluorene	230	NA	NA
Naphthalene	140	NA	NA
Phenol	160	NA	NA
Photomirex	7.38	NA	NA
Pyrene	790	NA	NA
TOTAL CARCINOGENIC FACTOR =			5,403

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-12

SCORES FOR CHEMICALS DETECTED IN MFLBC FISH SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	6150	2.0E-04	30,750,000
Photomirex	390	1.3E-03	312,000
Endrin	49	3.0E-04	163,333
Acetone	820	1.0E-01	8,200
Benzoic Acid	3300	4.0E+00	825
Tetrachloroethene	7	1.0E-02	700
Phenol	380	6.0E-01	633
2-Butanone	57	6.0E-01	95
Toluene	16	2.0E-01	80
Ethylbenzene	5	1.0E-01	50
Xylene (total)	20	2.0E+00	10
Benzene	2	NA	NA
N-Nitrosodiphenylamine	1000	NA	NA
TOTAL NONCARCINOGENIC FACTOR =			31,235,927

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Mirex	6150	5.3E-01	3,260
N-Nitrosodiphenylamine	1000	4.9E-03	5
Tetrachloroethene	7	5.2E-02	0
Benzene	2	2.9E-02	0
2-Butanone	57	NA	NA
Acetone	820	NA	NA
Benzoic Acid	3300	NA	NA
Endrin	49	NA	NA
Ethylbenzene	5	NA	NA
Phenol	380	NA	NA
Photomirex	390	NA	NA
Toluene	16	NA	NA
Xylene (total)	20	NA	NA
TOTAL CARCINOGENIC FACTOR =			3,265

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor. These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

TABLE B-13

SCORES FOR CHEMICALS DETECTED IN MFLBC FLOOD PLAIN SAMPLES

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	RfD (mg/kg-d)	NONCARCINOGENIC FACTOR
Mirex	4540	2.0E-04	22,700,000
Photomirex	132	1.2E-03	107,317
TOTAL NONCARCINOGENIC FACTOR =			22,807,317

CHEMICAL	MAXIMUM CONCENTRATION (ug/kg)	SLOPE FACTOR 1/(mg/kg-d)	CARCINOGENIC FACTOR
Mirex	4540	5.3E-01	2,406
Photomirex	132	NA	NA
TOTAL CARCINOGENIC FACTOR =			2,406

NOTE: Chemicals above the horizontal line drawn in the table indicates those chemicals with a factor of more than 1% of the total factor These chemicals are retained in the risk assessment.

NA means that chemical does not have relevant toxicity information and therefore cannot be scored.

APPENDIX C

Consideration of Tentatively Identified Compounds (TICs)

APPENDIX C

Consideration of Tentatively Identified Compounds (TICs)

Tentatively identified compounds (TICs) were reported for all media except flood plain soil, for which the only analytes quantified were mirex and photomirex. The lists of TICs detected in each medium are reported in Table C-1 and the number in each medium are summarized below.

Medium	Number of Reported TICs
On-site Test Pit Samples	40
On-site Pond Borings	49
Ground Water Samples	256
Air Samples	13
On-site sediments	121
Off-site Soil Boring Samples	17
Crane-Deming Soil	10
Railroad Track Test Pit Samples	22
MFLBC Surface Water Samples	4
MFLBC Sediment Samples	22
MFLBC Fish Tissue Samples	49

A discussion of TICs by medium and the likelihood that the TICs would significantly contribute to risk estimates developed in this risk assessment is provided in the following sections. In any assessment of TICs, it is important to recognize that the assigned identity of a TIC is, in most cases, highly uncertain. Further, estimates of concentrations of TICs are highly uncertain and could be orders of magnitude higher or lower than the actual concentration (see USEPA 1989, p. 5-18). Recognizing these uncertainties in the available data for TICs, the following preliminary conclusions can be reached.

1. On-site Test Pit

Forty TICs were tentatively identified in on-site test pit samples. Several of these TICs are characterized by general chemical class (e.g., alcohol, aldehydes, alkanes, and alkene). Although the structures of these TICs are not characterized sufficiently to allow an assessment of potential risk, they appear to be relatively simple molecules and would be expected to degrade rapidly. A number of the TICs tentatively identified in test pit samples are halogenated organic compounds and other aromatic compounds that would not be expected to occur naturally. Analyses for chemicals on the Target Analyte List/Target Compound List (TAL/TCL, or target list compounds) also identified a number of halogenated and substituted aromatic compounds. The concentration and toxicity screen, however, suggests that the relative risk associated with mirex in test pit samples far exceeds the risks associated with other target list compounds. It is, therefore, unlikely that TICs would significantly contribute to overall risk estimates quantified for test pit samples.

2. On-site Pond Borings

Forty-nine TICs were reported in on-site pond boring samples. A number of these TICs are halogenated organic compounds and other aromatic compounds that would not be expected to occur naturally. Of the target list compounds, a number of chlorinated organic compounds were detected with maximum concentration as high as several hundred to several thousand ppm. Therefore, TICs are unlikely to significantly contribute to risk estimates quantified for on-site pond boring samples.

3. Ground Water

Two hundred fifty-six (256) TICs were tentatively identified in ground water. The majority of these TICs (greater than 200) were not characterized sufficiently to permit an assessment of potential risk (e.g., "mix aniline + chloroaliphatic," "mix chlorinated aliphatic," "unknown aliphatic hydro + silane"). A number of reported TICs tentatively identified in ground water were chlorinated compounds, a benzenesulfonamide mixture or derivative, or a substituted aromatic compound. The TAL/TCL analysis also identified a number of chlorinated hydrocarbon compounds, benzene, and various substituted aromatic compounds, which pose a relatively high potential health risk. Therefore, it is unlikely that risks posed by the TICs in ground water would result in a significantly different conclusions regarding potential risks associated with ground water.

4. Air

Thirteen volatile compounds were tentatively identified in air. Among these are five halogenated compounds, benzene, other hydrocarbons, and sulfur dioxide. Because of the substantial uncertainty about both the identities and concentrations of these TICs, it is not possible to assess whether or not these TICs would significantly contribute to overall risk associated with chemicals in on-site air.

5. On-site Sediments

One hundred twenty-one TICs were tentatively identified in on-site sediments. As in other on-site media, the TICs included a number of halogenated hydrocarbons, various tentatively identified substituted aromatic compounds, and many unknown hydrocarbons mixtures. Also among the list of TICs in on-site sediments are compounds that are likely to be naturally-occurring components of biological systems (e.g., cholest-5-en-3-ol, carboxylic acids, an unknown steroid, and an unknown amine). Analyses for target list compounds showed a number of chlorinated compounds, pesticides, PAHs, and other aromatic compounds, with maximum concentrations in the ppm range. It is unlikely, therefore, that any risks posed by TICs in on-site sediments would result in significantly different conclusions regarding potential risks for on-site sediments.

6. Off-site Soil Borings

The majority of the 17 TICs in off-site soil boring samples are naturally-occurring organic compounds, including fatty acids, steroids, a plant sterol, and hydrocarbon chains (alkanes). Two polynuclear aromatic hydrocarbons (PAHs), both substituted naphthalenes, were tentatively identified, but are unlikely to contribute significantly to the risk associated with other noncarcinogenic PAHs detected in off-site soil borings that were on the TCL/TAL list. Benzeneacetic acid and 2H-1-benzopyran-2-one were also reported in off-site soil samples. Benzeneacetic acid is a relatively simple compound and would likely undergo relatively rapid degradation in the environment. The toxicological potential of 2H-1-benzopyran-2-one is uncertain, although the structure in not inconsistent with certain biological compounds.

One halogenated aromatic hydrocarbon was tentatively identified in off-site soil borings; this TIC was not sufficiently identified to permit any assessment of potential risk.

7. Crane-Deming Soil

The majority of the 10 TICs tentatively identified in Crane-Deming soil samples were characterized as an unknown hydrocarbon (carboxyl, carboxylic acid, aliphatic alcohol, aliphatic hydrocarbon, aliphatic/aromatic, or aromatic compound). These are likely to be relatively simple molecules that would undergo relatively rapid degradation in the environment.

One chlorinated hydrocarbon TIC and one PAH were tentatively identified, although the identifications were not sufficient to allow any assessment of risk.

In general, based on the nature of TICs present in Crane-Deming samples, it is unlikely that these TICs would significantly contribute to overall risks associated with Crane-Deming soil.

8. Railroad Track Test Pit

Twenty-two TICs were detected in railroad track test pit samples, including four chlorinated organic compounds and a number of PAHs. Chlorinated organic compounds and PAHs, in addition to mirex, photomirex, and kepone, are the predominant target compound list constituents in railroad track test pit samples. Any additional risks associated with TICs in railroad test pit samples are not likely to result in significantly different overall conclusions regarding potential risks.

9. MFLBC Surface Water

Only four TICs were tentatively identified in surface water. Two of the TICs were described as unknown compounds containing oxygen or alcohol groups. This chemical characterization is insufficient to permit any assessment of potential risk. A third TIC detected in surface water samples was an octadecene isomer, which is a straight chain aliphatic. It is likely that this compound readily degrades and therefore presents little risk to potentially exposed populations.

The only other TIC of note in surface water was identified as "ethane, 1,1,2-trichloro-1,2." This nomenclature, however, is chemically meaningless. This substance was reported in only a single sediment sample at a low concentration, and 1,1,2-trichloroethane was not detected in that sample. Given the low level tentatively measured, and the question about the identity of the substance, no meaningful risk can be associated with this substance.

10. MFLBC Sediment

The majority of the 22 TICs in sediment appear to be natural components of biological systems (fatty acids and their esters, other hydrocarbons, cholesterol and other steroids, and vitamin E). Because these are normal nutrients, they present no risk to potentially exposed populations. One PAH was tentatively identified as benzo(e)pyrene. This PAH was detected in a single sample (SD-30) at a very low level (260 ppb), and is not likely to add significantly to the risk from other PAHs that have been detected in sediment.

Several TICs (alkanes, alkenes) were not identified sufficiently to permit any assessment. These classes of compounds, however, are relatively simple molecules that would be expected to degrade rapidly. Rapid degradation would also be likely for benzeneacetic acid; phosphoric acid, triphenyl ester; and phenol, 2,6-bis(1,1-dimethylethyl). The latter is similar in structure to the natural flavor ingredient thymol (phenol, 2-(1,1-dimethylethyl)-5-methyl), found in thyme and mint, and probably represents a natural phenol from plants.

Also detected in sediment was a TIC identified as "ethane, 1,1,2-trichloroethane-1,2,2." As discussed above for TICs in surface water, this nomenclature is chemically meaningless. Similar to surface water, the constituent was only detected in one sample at a very low concentration and 1,1,2-trichloroethane was not detected in that sample.

11. MFLBC Fish

The vast majority of the 49 TICs detected in fish tissue appear to be natural components of biological systems, mainly fatty acids and related substances (fatty acid esters, fatty acid aldehydes), other hydrocarbons, indole (a degradation product of the amino acid tryptophan), and cholesterol and other steroids. As described above, they would not be expected to present any risk to a potentially exposed individual. As in sediment samples, some TICs are not identified sufficiently (e.g., "aldehyde," "alkene," "nitrile") to permit any assessment. All of these, however, appear to be relatively simple molecules, have formulas consistent with those of components of biological systems, and would be expected to degrade rapidly.

12. Conclusions

Samples of on-site soils, sediment, ground water, and air and railroad track test pits contain various halogenated organic compounds, PAHs and other aromatic

compounds that are not likely to be components of natural biological systems. In all cases, analysis of target list compounds in the same medium show a similar spectrum of halogenated and aromatic compounds to be present. Therefore, a quantitative assessment of potential risks associated with these TICs, even if one could be performed, would be unlikely to change the overall conclusions regarding potential risk associated with chemicals in Site media. Other compounds tentatively identified in on-site and railroad track test pit samples are relatively simple compounds (alkanes, alkenes) that would be expected to degrade rapidly. Some TICs in the sampled media, and ground water in particular, were not identified sufficiently to permit an assessment of potential risk.

The majority of TICs in off-site soil borings and soil samples from Crane-Deming are naturally-occurring compounds or, like benzeneacetic acid, are relatively simple compounds that would likely degrade rapidly in the environment. Only two halogenated hydrocarbons and three PAHs were tentatively identified in these off-site soils.

The vast majority of the TICs detected in fish, sediment, and surface water are natural components of biological systems and would not be expected to present any risk to potentially exposed populations. A few additional TICs are either present only in one or a few samples at low concentrations (e.g., benzo(e)pyrene) or are simple molecules that are likely to degrade readily (e.g., benzeneacetic acid) and, as such, are not likely to add significantly to risk. The remaining TICs are insufficiently identified to permit any assessment; however, most of these appear to be relatively simple molecules that would be expected to degrade rapidly.

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REFERENCES

U.S. Environmental Protection Agency (USEPA). 1989a. *Risk assessment guidance for Superfund. Volume I: Human health evaluation manual.* Office of Emergency and Remedial Response. EPA/540/1-89/002.

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TABLE C-1
Tentatively Identified Compounds

ON-SITE TEST PIT SAMPLES

Volatile Compounds

4-Octene, (Z)

Ethyl Methyl Benzene Isomer

Pentane, 2,3-Dimethyl

Semivolatile Compounds

1-Chloro-4-(phenylsulfonyl)-benzene

1,1'-Sulfonylbis-benzene

1,4-bis(4-Bromophenyl)-1,4-Butanedione

1,5-Diphenyl-1,4-pentadien-3-one

2-Bromo-1,3-cyclopentanedione

2-Propanol, 1,3-dichloropropane

2-(2-Hydroxyethyl)-1H-Isoindole-1,3(2H)-dione

4-Chlorobenzene Sulfonamide

4-Phenyl-3-buten-2-one

Adipate

Alcohol

Aldehydes

Alkanes

Alkene

Alpha-oxo-benzenacetonitrile

Benzene-dimethoxyethyl Isomer

Benzenesulfonamide

Benzene, (1,2-Dimethoxy)ethyl

Benzophenone

Benzoyl Chloride, 4-Bromo

Bis(bromophenyl)diazene Isomer

TABLE C-1 Tentatively Identified Compounds
Bromochlorobenzene Isomer
Diazene, bis(3,4-dichlorophenyl)
Diazene, bis(3,4-dichlorophenyl)1-oxide
Dichlorophenol Isomer
Diphenylethanedione
Eicosane
Heptacosane
Heptadecane
Hexadecane
Hexadecanoic Acid
Mirex
Octadecanoic Acid Ester
Octane Isomer
Pentadecane
Phenol, 2,4-dichloro-, benzene sulfonate
Tridecane
ON-SITE POND BORINGS
Volatile Compounds
2-Propanol
3-Methylpentane
Alkene
Benzene, (1,2-dimethoxyethyl)-
Ethane, 1,1,2-Trichloro-1,2,2
Methylethylbenzene Isomer
Semivolatile Compounds
1-Phenyl-1,2-ethanediol
1-(4-Nitrophenyl)-3-phenyl-2-propen-1-one

TABLE C-1
Tentatively Identified Compounds

1,3-Cyclopentadiene, 1,2,3,4-Tetrachloro-
3,4-Dichlorostyrene
Benzaldehyde
Benzaldehyde (ACN)(DOT)
Benzenamine, Dichloro- Isomer
Benzenamine, Dimethylnitro- Isomer
Benzenemethanol, alpha-(Chloroethyl)
Benzenesulfonamide
Benzenesulfonamide, 4-Methyl
Benzenesulfonamide, alpha-(chloromethyl)-
Benzenesulfonyl Azide
Benzene, 1-Chloro-4-(1,2-Dichloroethyl)-
Benzene, 2,4-Dibromo-1-Methoxy-
Benzene, 2,4-Dichloro-, Benzenesulfonate
Benzene, (1-Methylethyl)-
Benzene, (1,2-Dichloroethyl)-
Benzene, (1,2-Dimethoxyethyl)
Benzene, (Chloroethenyl) - Isomer
Benzophenone
Bromophenol Isomer
Butanoic Acid
Diazene, Bis(2-Bromophenyl)-
Diazene, Bis(3,4-dichlorophenyl),1-oxide
Diazene, Bis(Bromophenyl)- Isomer
Dimethylnitrobenzenamine Isomer
Ethanedione, Diphenyl-
Ethanone, 1-Phenyl
Ethanone, 1-(3-Nitrophenyl)-
Ethanone, 1-(4-Nitrophenyl)-

TABLE C-1 Tentatively Identified Compounds
Ethanone, 1-(Aminophenyl) - Isomer
Ethanone, 1-(Nitrophenyl)-
Hexadecanoic Acid
Methanone, (4-Bromophenyl)phenyl
Octadecanoic Acid, 2-Methylpropyl Ester
Octadecanoic Acid, Ester
Pentachloroethane
Phenol 2,4-dichloro-, Benzenesulfonate
Phenol, 2,6-bis(1,1-Dimethylethyl)-4-Methyl
Substituted Benzene
Substituted Phenol
[1,1'-Biphenyl]-2-ol
GROUND WATER
Volatile Compounds
1-Chlorocyclopentene
2-Chloropropane
3-Methylpentane
Methylcyclopentane
Semivolatile Compounds
1H-indole-3-ethanol, 5-hydro
1-Phenylethanone + Unknown
1,1'-Biphenyl-2-OL
1,1-Sulfonylbisbenzene
1,2-Cyclohexanediol
1,2-Ethanediol, 1-Phenyl
1,4-Pentadiene-3-one, 1,5-Diphen
2-Cyclohexen-1-one

TABLE C-1
Tentatively Identified Compounds

2-Propanol, 1,3-dichloro-
2-Propen-1-one, 1-(Nitrophenyl)
2-Trifluorormet Benzamine + Unknown
2,4-Imidazolidinedione,5,5-
3-Buten-2-one, 4-Phenyl-
4-Benzoylmorpholine
5,5-Diph-2,4-Imidazolidinedione
Benyoyl Glycine + Unknown
Benzamide
Benzenamine, 2-(Trifluoromet)
Benzenamine, 3-(Trifluoromet)
Benzenesulfonamide
Benzenesulfonamide Unknown Chlor
Benzenesulfonamide + Unknown
Benzenesulfonamide + Unknown Aromatic
Benzenesulfonamide + Unknown Nitroarom
Benzenesulfonamide, 4-Chloro
Benzenesulfonamide, 4-Methyl
Benzenesulfonamide, 4-Methyl + Unknown
Benzenesulfonamide, N-Phenyl
Benzene, 1-Nitro-3-(Trifluor)
Benzene, 1,1'-Sulfonylbis
Benzensulfonamide, 2-methyl
Benzoic Acid
Benzoic Acid + Mix Unknown
Benzoic Acid + Unknown Chlorphene
Benzoic Acid, 2-Bromo-
Benzonitrile
Benzonitrole

TABLE C-1
Tentatively Identified Compounds

Benzophenone
Bromobenzoic Acid + Unknown
Carboxylic Acid
Chlorinated Alkoxybiphenyl
Chlorobenzenesulfonamide
Chlorobenzenesulfonamide + Unknown
Chloroethenylbenzene + Unknown
Chloromethylsulfonylbenzene
Cycloalkyl/Amine/Cyclohex.
Cyclopentanecarboxaldehyde
Decanoic Acid
Dibromobenzoic Acid
Diphenylmethanone
Diphenylmethanone + Unknown
Dodecanamide, N,N-Bis(2-Hydr)
Ethanedione, Diphenyl-
Ethane, Pentachloro-
Ethanone, 1-Phenyl-
Ethylmethylbenzenesulfonamide
Imidazolidinedione Unknown Subs
Methanone, Diphenyl-
Methylsulfonylbenzene
Mix Aliphatic Unknowns
Mix Analine + Chloroaliphatic
Mix Aromatic Species
Mix Aromatic Unknowns
Mix Aromatic + Unknowns
Mix Benyoyl + Hydroxybenzoic Acid
Mix Benzenesulfonamide/Chlor

TABLE C-1
Tentatively Identified Compounds

Mix Benzoic Acid + Unk Aliphatic
Mix Benzoic Acid + Unknowns
Mix Benzoyl + Unknown
Mix Benzoyl + Unknown Alkoybenzene
Mix Benzsulfonamide/Unknown
Mix Bromoalkyl + Unknowns
Mix Bromobenzoic + Nitrophenet
Mix Chlorinated Aliphatic
Mix Chlorinated Aromatic + Unknown
Mix Chlorobenzoic Acid + Unknown
Mix Cyanobenzene + Unknowns
Mix N-Ethyl-4-methylbenzulfa + Unknown
Mix Nitrobenzoic Acid + Chlbens
Mix Pentachloroethane + Unknown
Mix Sub Benzenesulfonamide & Silane
Mix Sulfonated Phenyl Naphth
Mix Unknown Aromatic + Unknown
Mix Unks.
Mix Unknown Alipha Hydro + Silane
Mix Unknown Aliphatic Hydro & Silane
Mix Unknown Aliphatic & Silane
Mix Unknown Aliphatics
Mix Unknown Alkylbenzene
Mix Unknown Aromatic Amine
Mix Unknown Aromatic + Unknown Aliphatic
Mix Unknown Aromatics
Mix Unknown Benzenesulfonamide
Mix Unknown Benzoic Acid + Chlorop
Mix Unknown Benzyl Alcohol

TABLE C-1
Tentatively Identified Compounds

Mix Unknown Bromobenzoic Acid
Mix Unknown Cyclohexenol + Unknown
Mix Unknown Dichlorophenol + Benzo
Mix Unknown Fluorinated Aromatic
Mix Unknown Hydrocarb + Chlorohyd
Mix Unknown Nitrobenzeneamine + Unknown
Nitroaromatic Unknowns
Nitrobenzene + Benzoic Acid Mix
Phenol, 2,4-Dichloro-, Bensul
Phenol, 2,4-Dichloro-,Benzene
Phenyl Sub Amino Nathyene
Phenylethanone
Polychlorinated Aliph.
S-Phen Ester Benzsulfonothio
Sulfur, Mol. (S8)
Trichlorobenzene
Trifluorometbenzenamine + Unknown
Unknown
Unknown (MWT. 116)
Unknown 1MS Derivative
Unknown 4-Trifluoromet Benzenamine
Unknown Acetyl
Unknown Acid
Unknown Aliphatic
Unknown Aliphatic Acid
Unknown Aliphatic Alcohol
Unknown Aliphatic Amine
Unknown Aliphatic Chlorinated
Unknown Aliphatic Hydro + Silane

TABLE C-1
Tentatively Identified Compounds

Unknown Aliphatic Hydrocarb
Unknown Aliphatic Polychlorinat
Unknown Aliphatic + Silane
Unknown Aliph. Hydrocarb. + Silane
Unknown Alkoxy Ethanol
Unknown Alkoxy Sub. Aliphatic Alc
Unknown Alkoxybenzene
Unknown Alkoxyethanol
Unknown Alkylbenzene
Unknown Alkylbenzene Alkoxy Sub
Unknown Alkylpyridine
Unknown Aloxybenzene
Unknown Aloxyethanol
Unknown Amine
Unknown Amino Benzoic Acid
Unknown Aminophenylethanone
Unknown Aromatic
Unknown Aromatic Acid
Unknown Aromatic Alcohol
Unknown Aromatic Amine
Unknown Aromatic Amine + Unknown
Unknown Benzenamine/Unknown Substit
Unknown Benzene Sulfonated
Unknown Benzeneamine
Unknown Benzeneamine/Unknown Subs
Unknown Benzenediol
Unknown Benzenesulfonamide + Aroma
Unknown Benzenesulfonamide + Nitroben
Unknown Benzenesulfonamide + Unknown

TABLE C-1
Tentatively Identified Compounds

Unknown Benzofuran + Unknown Benzoyl
Unknown Benzoic Acid
Unknown Benzoyl
Unknown Benzoyl + Unknown
Unknown Benzyl Alcohol
Unknown Biphenyl
Unknown Biphenyl Substituted
Unknown Biphenylol
Unknown Brominated Aliphatic
Unknown Bromoaliphatic
Unknown Bromoalkyl
Unknown Bromoaromatic
Unknown Bromobenzoic Acid
Unknown Bromobenzoic Acid + Aromatic
Unknown Bromodichlorobenzene
Unknown C_6 Cyclic
Unknown Carb Acid
Unknown Carboxylic Acid
Unknown Chlor Naphtaylene/Biphen
Unknown Chlorinated Alhyl
Unknown Chlorinated Alkylbenzene
Unknown Chlorinated Aromatic
Unknown Chlorinated Biphenyl
Unknown Chlorinated Biphenylol
Unknown Chlorinated Napthylene
Unknown Chlorinatedcyclohexanone
Unknown Chloroalkanol
Unknown Chloroalkyl-Aromatic
Unknown Chloroaromatic

TABLE C-1
Tentatively Identified Compounds

Unknown Chlorobenoyl
Unknown Chlorobenzenesulfonamide
Unknown Chlorobenzoic Acid
Unknown Chlorobiphenyl
Unknown Chlorobiphenylol
Unknown Chlorocyclohexanol
Unknown Chlorocyclohexanone
Unknown Chloroethenylbenzene
Unknown Chloronitroaromatic
Unknown Cyclic Alcohol
Unknown Cyclic Hydrocarb/Unknown Sub.
Unknown Cycloalkanol
Unknown Cyclohexandiol
Unknown Cyclohexanol
Unknown Cyclohexanol Subs
Unknown Cyclohexenol
Unknown Cyclohexenol + Unknown Mix
Unknown Cyclohexyl
Unknown Cyclopentanol Subs.
Unknown Cyclopenteneol
Unknown Dibromoaromatic
Unknown Dibromobenzene
Unknown Dibromobenzoic Acid
Unknown Dichlorinated Ali.
Unknown Dichloroalkanol
Unknown Dichloroaromatic
Unknown Dichlorobiphenyl
Unknown Dichlorophenol
Unknown Dihydroxy Sulfonate Arom.

TABLE C-1
Tentatively Identified Compounds

Unknown Dimethylpyridine
Unknown Halogenated Aliphatic
Unknown Imadazolidindione
Unknown Methbenzenesulfonic Acid
Unknown Methoxybenzene
Unknown Methylnitrobenzene Amine
Unknown Mix Benzenesulfonyl
Unknown Mix Trimeth Phenol/Aroma
Unknown Nitro Phenyl
Unknown Nitroaniline
Unknown Nitroaromatic
Unknown Nitrobenzenamine
Unknown Nitrobenzene
Unknown Nitrofluorobenzene
Unknown Nitrogen Containing
Unknown Nitrogen Heterocyclic
Unknown N-Phenylsulfonyl/Bensulf
Unknown Phenyl
Unknown Phenylsulfonylbenxsulfon
Unknown Phenylsulfonylnaphtal
Unknown Polychlorinated Aliphatic
Unknown Polyhalogenated Aliphatic
Unknown Subst Bromobenzoic Acid
Unknown Subst. Benzenesulfonamide
Unknown Subst. Benzyl Alcohol
Unknown Subst. Biphenyl
Unknown Subst. Bromobenzene
Unknown Subst. Chlorinated Benzene
Unknown Subst. Morpholine

TABLE C-1 Tentatively Identified Compounds
Unknown Subst. Phenylbutenone
Unknown Subs. Cyclohexane
Unknown Sub. Morpholine
Unknown Sulfonated Aromatic
Unknown Sulfonated Benzene
Unknown Sulfonylbenzene
Unknown Thioaromatic
Unknown Trichloroaliphatic
Unknown Trifluoromethbenzenamine
Unknown Trifluoromethyl Analine
Unknown Triphenylphosph Com
Unknown Triphenylphosphine
AIR SAMPLES
Volatile Compounds
1,1,2-Trichlorotrifluoroethane
1,4-Dioxane
Acetone
Benzaldehyde
Benzene
Butane
Chlorodifluoroethane
Dichlorodifluoromethane
Hexane
Methylene Chloride
Phenylethanone
Sulfur Dioxide
Trichlorofluoromethane

TABLE C-1
Tentatively Identified Compounds

ON-SITE SEDIMENT SAMPLES

Volatile Compounds

Mix Dichloroalkyl + Carboxyl

Unknown Aliphatic

Unknown Alkyl

Unknown Alkyl Mix

Unknown Alkyl/Carboxyl Mix

Semivolatile Compounds

9H-Xanthen-9-one

Aliphatic Hydrocarb. Phthal.

Aliphatic Hydrocarb/Silyl

Benzenesulfonamide

Benzene, 1,1'-Sulfonylbis-

Bromobenzene Unknown

Bromoethylbenzene Unknown

Bromophenyl Unknown

Carboxylic Acid Unknown/Sulfur

Cholest-5-en-3-ol

Chlorobromo Unknown

Chlorodibromo Unknown

Chloronapthylamine MWT. 204

Chlorosulfonyl Benzene Unknown

Cyclohexane Unknown

Cyclohexanol Unknown

Diazene, Bis(3,4-Dichlorophe

Dibromobiphenyl Unknown MW-340

TABLE C-1
Tentatively Identified Compounds

Dibromodichlorobenzoic Acid
Dibromophenyl Unknown
Methanone, Diphenyl
Mix Aliphatic Hydrocarb. Unknown
Mix Unknowns
Mix Unknown Aliphatics
Mix Unknown Alkyls, Sulf
Mix Unknown/Aromatics + Aliphatics
Mix-57-10-3 + Sulfur
Mix-Aliphatic Hydrocarb/Sil
Mix-Aromatic Unknown, Phthal.
Mix-Bromophenyl Unknown/Other Unknown
Mix-Carboxylic Acid Unknown/Sil.
Mix-Carboxylic Acid/Hydrocarb.
Mix-Chlorodibromoaliphatic
Mix-Dichlorohydrocarb/Silyl
Mix-Halogenated Unknown
Mix-Molecular Sulfur/Unknown
Mix-Phthalate + Other Unknown
Mix-Silyl, Phthalate, Steroid
Mix-Silyl, Unknown Steroid
Mix-Unknowns
Mix-Unks. MW-410 Aliph/Sil
Mix-Unks., HC, Phthal, Si
Mix-Unknown
Mix-Unknown Aliphatic Hydrocarb.
Mix-Unknown Aliphatics/Silyl
Mix-Unknown Biphenyl + Sulfur
Mix-Unknown Carboxylic Acid

TABLE C-1
Tentatively Identified Compounds

Mix-Unknown Hydrocarb/Silyl
Mix-Unknown RBR3CL Aliphatic
Mix-Unknown + Silyl
Mix. Unknown Aliphatics
MW-116 Chlorohydrocarb. Unknown
MW-257 Unknown Aliphatic N
MW-262 Chloroaromatic Biphen.
MW-396 Napthylamine Unknown
Napthylamine Unknown
Sulfur, MW. (S8) + Unknown
Tetradecanoic Acid, Ester Unknown
Tetramethyldiphenylsulfone
Unknown
Unknown Aliphatic Amine
Unknown Aliphatic Hydrocarb.
Unknown Alipahtic/Amine
Unknown Aliphatic
Unknown Aliphatic Ester
Unknown Aliphatic Hydrocarb
Unknown Aliphatic MW 423 + Si
Unknown Aliphatic + Silyl
Unknown Aliphatic + Sulfur
Unknown Aliphatics
Unknown Aliphatic, Phthalate, Si
Unknown Aliphatic/Amine
Unknown Aliphatic/Silyl
Unknown Alkenal
Unknown Alkylamine + Unknown
Unknown Alkylbenzene

TABLE C-1
Tentatively Identified Compounds

Unknown Amine
Unknown AR, Phenyl, MW-358
Unknown Benzoyl + Sulfur
Unknown Biphenyl-ol
Unknown Bromodichloro
Unknown Bromophenyl MW-302
Unknown Bromophenyl MWT-183
Unknown C12H9OCL Aromatic
Unknown Carboxylic Acid
Unknown Carboxylic Acid Ester
Unknown Carboxylic Acid, Sul
Unknown Chloraliphatic/Unknown
Unknown Chlorinated Aliphatic
Unknown Chloroaliphatic
Unknown Chloronapthalene
Unknown Hydrocarb.
Unknown Hydrocarb. Aliphatic
Unknown Hydrocarb. MW-104
Unknown Hydrocarb/Silyl
Unknown Isomer of RT 29.55
Unknown Ketone/Aldehyde
Unknown Mix Aliphatics
Unknown Napthalene/Sulfur
Unknown Napthalene/Unknown
Unknown Napthlamine/Silyl
Unknown Nitroaniline
Unknown Nitrobenzene
Unknown PAH, MW 252 + Silyl
Unknown Phenyl

TABLE C-1 Tentatively Identified Compounds
Unknown Phthalate
Unknown Polychlorinated
Unknown Polychlorinated Ali.
Unknown Polyhalogenated Ali.
Unknown Polyhalogenated Hyd-Carb
Unknown Polyhalogenated Mix
Unknown Polyhalogenated (Br)
Unknown Poss. Steroid
Unknown Steroid
Unknown Sub. Phenyl/Silyl
Unknown Tribromo. Phenyl, MW-402
OFF-SITE SOIL BORING SAMPLES
Semivolatile Compounds
1-Methyl Naphthalene
2H-1-Benzopyran-2-one
9-Hexadecenoic Acid
Alkanoic Acid
Benzeneacetic Acid
Cholest-5-en-3-one
Cholestane Isomer
Dimethyl Naphthalene Isomer
Dodecane
Eicosene Isomer
Halogenated Aromatic
Hexadecanoic Acid
Octadecanoic Acid
Octadecanoic Acid Isomer
Sitosterol Isomer

TABLE C-1 Tentatively Identified Compounds
Tetradecanoic Acid
Tridecane
CRANE-DEMMING SAMPLES
Volatile Compounds
Unknown
Unknown Carboxyl
Unknown Dichlorocarboxyl
Unknown Hydrocarbon
Semivolatile Compounds
Unknown Aliphatic Alcohol
Unknown Aliphatic Hydrocarbon
Unknown Aliphatic/Aromatic
Unknown Aromatic Hydrocarbon
Unknown Carboxylic Acid
Unknown PAH
RAILROAD TRACK TEST PIT SAMPLES
Semivolatile Compounds
1-Methylnaphthalene
1-(1,1-Dimethyl)-2-methyl-1-3-propane Propanoic Acid
1-(4-Nitrophenyl)-Ethanone Isomer
1,2-Dimethyloxyethyl Benzene
2,4-Dichloro-benzenesulfonate Phenol
2,6,10,14-Tetramethyl Pentadecane
4-Chlorobenzene Sulfonamide
Adipate

TABLE C-1 Tentatively Identified Compounds
Alkanes
Benzenemethanol, alpha-(chloromethyl)-
Benzenesulfonamide
Benzene, (1,2-dimethoxyethyl-)
Benzo(e)pyrene
Bromodichlorobenzene Isomer
Dimethylnaphthalene Isomer
Ethanedione, Diphenyl-
Ethanone, 1-phenyl
Methylanthracene/Phenanthrene Isomer
Methylphenanthrene Isomer
Naphthalene, -Dimethyl Isomers
Naphthalene, -Trimethyl Isomers
S-S'[Thiobis(methylene)]ester Ethane Thioic Acid
MFLBC SURFACE WATER SAMPLES
Volatile Compounds
Ethane, 1,1,2-Trichloro-1,2
Semivolatile Compounds
Octadecene Isomer
Unknown (unsaturated or alcohol)
Unknown oxygen compound
MFLBC SEDIMENT
Volatile Compounds
Ethane, 1,1,2-Trichloro-1,2,2

TABLE C-1 Tentatively Identified Compounds
Semivolatile Compounds
9-Hexadecenoic Acid
9-Octadecenoic Acid
Alkane
Alkanoic Acid
Alkene
Benzeneacetic Acid
Benzo(e)pyrene
Cholesterol
Heptadecenoic Acid
Hexadecanoic Acid
Hexadecanoic Acid, Methyl Ester
Octadecanoic Acid
Octadecanoic Acid, 2-Methylpropyl
Pentacosane Isomer
Pentadecanoic Acid
Phenol, 2,6-Bis(1,1-Dimethylethyl)-
Phosphoric Acid, Triphenyl Ester
Sulfur
Tetradecanoic Acid
Tetradecanoic Acid, Hexadecyl
Vitamin E
MFLBC FISH TISSUE
Semivolatile Compounds
11-Eicosenoic Acid, Methyl Ethyl-
1H-Indole
9-Hexadecanoic Acid

TABLE C-1
Tentatively Identified Compounds

9-Hexadecenoic Acid, Methyl-
9-Hexadecenoic Acid, Methyl Ester
9-Octadecanoic Acid, 12-(Acetyloxy)-
9-Octadecenoic Acid Z-Ether
Aldehyde
Alkene
Alkenoic Acid
Cholesterol
Cholestnone Isomer
Decyne Isomer
Dodecanoic Acid
Heptadecane
Heptadecanoic Acid
Heptadecanoic Acid Alkene
Heptadecanoic Acid Isomer
Heptadecanoic Acid, 16-Methyl
Heptadecanoic Acid, Methyl-
Heptadecanoic Acid, Methyl Ethyl-
Heptadecanoic Acid, Substitute
Heptadecenoic Acid
Heptadecenoic Acid Isomer
Hexadecanal (ACN)
Hexadecanoic Acid
Hexadecanoic Acid, Substitute
Hexadecenoic Acid Alkene
Hexadecenoic Acid Isomer
Hexadecenoic Acid, Methyl Ester
Hexadecenoic Acid, Methyl Ethyl-
Hexadecenoic Acid, Methyl Ethyl Isomer

TABLE C-1
Tentatively Identified Compounds

Nitrile
Octadecadienoic Acid
Octadecadienoic Acid, Methyl
Octadecanal
Octadecanoic Acid
Octadecanoic Acid, Methyl Ester
Octadecenoic Acid Isomer
Octadecenoic Acid, Methyl Ester
Octadecenoic Acid, Methyl Ethyl
Pentadecanoic Acid
Pentadecanoic Acid, 14-Methyl-
Pentadecanoic Acid, Methyl-
Propanoic Acid, 2-Methyl-
Tetradecanoic Acid
Tetradecanoic Acid, Methyl-
Tetradecanoic Acid, Methyl Ester
Undecane Isomer

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APPENDIX D

A Review of Mirex/Technical Support for Evaluating the Carcinogenic Potential of Mirex

A Review of Mirex

A REVIEW OF MIREX

May 12, 1992

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I. INTRODUCTION

Mirex is a fully chlorinated organic compound having the molecular formula $C_{10}Cl_{12}$. It is not known to occur naturally. Mirex was first synthesized in the mid 1940s, but was not marketed commercially in the United States until the late 1950s.

PRODUCTION AND USE

The first patents were granted for this compound in the mid-1950s and claimed its use as an insecticide and as an intermediate in carrying out chemical reactions. Subsequently, many different uses and properties of mirex were patented in several countries by a number of different companies. The name mirex typically refers to the pure technical product or to the various pesticide baits and formulations in which it was used.

Under the name dechlorane, mirex was also marketed by the Hooker Chemical Company in the United States from 1959-1972 as a stable, fire-retardant additive for use in thermoplastic, thermosetting, and elastomeric resin systems. Reportedly, it was also useful in paper, paint, rubber, electrical, adhesive, and textile applications (IARC 1979, Kaiser 1978). The use of mirex as a flame retardant generally exceeded its various other uses.

REGULATORY HISTORY

In 1961 Hooker Chemical Company was granted a registration (administered first by the U.S. Department of Agriculture and subsequently by the U.S. Environmental Protection Agency) for production of technical-grade mirex. This product was intended for use only in formulating pesticide products. Subsequently, 10 registrations for a variety of mirex-containing pesticide formulations were granted to Allied Chemical Corporation.

During the 1960s mirex was the pesticide of choice in a Department of Agriculture program to eradicate the imported fire ant -- a widespread pest prevalent in the Southeastern United States. By 1971, however, the U.S. Environmental Protection Agency (USEPA) issued a notice of cancellation of all mirex registrations because of evidence from an animal bioassay of potential carcinogenicity (Innes et al. 1969). Gradually, over the next several years, various uses and methods of application for mirex were phased out. By the end of 1977, pursuant to the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the USEPA announced its intent to cancel all registrations for products containing mirex as an active ingredient. Conditions for carrying out the order and provisions for the use of existing

stocks of mirex products were described at that time. With minor exceptions, use of mirex-containing pesticides was effectively prohibited by mid-1978. From 1962 to 1976 about 226,000 kg of mirex were applied to 132,000,000 acres in 10 states (IARC 1979).

In 1973 the USEPA established tolerances for residues of mirex in food products (38 FR 12215). These values were as follows:

> 0.1 parts per million (ppm) for the fat of meat from cattle, goats, hogs, horses, poultry, and sheep;
>
> 0.1 ppm for milk fat and eggs; and,
>
> 0.01 ppm for all other raw agricultural commodities.

For purposes of conducting residue monitoring programs, the Food Safety and Inspection Service (FSIS) of the U.S. Department of Agriculture (USDA) also used an action level of 0.1 ppm for the fat of meat and meat byproducts from cattle, goats, swine, horses, sheep, poultry, and rabbits (50 FR 11185). In 1978, based on a recommendation made by the USEPA, the FDA established an action level of 0.1 ppm for residues of mirex in fish (43 FR 14736).

In 1986, the USEPA revoked all of its existing tolerances for mirex for the agricultural commodities listed above (51 FR 45114). The USEPA noted that these tolerances were not revoked concurrently with the cancellation of the pesticide registrations for mirex because of the compound's slow rate of degradation and its persistence in the environment. Based on monitoring data available at the time, the tolerances for mirex were revoked, and the USEPA recommended that FDA retain its existing action level of 0.1 ppm for residues of mirex in fish. In addition, USEPA recommended that the FSIS retain its existing action level of 0.1 ppm for residues of mirex in the fat of meat and meat byproducts of livestock. Because mirex residues had not been reported in other raw agricultural commodities, USEPA recommended that no new action levels be established by FSIS to replace the USEPA tolerance of 0.1 ppm for mirex in milk fat and eggs and 0.01 ppm for residues in all other raw agricultural commodities.

Based on data for fresh- and salt-water invertebrates and on a variety of supplemental data, in 1976 the USEPA established an ambient water-quality criterion of 0.001 μg/l for both freshwater and marine aquatic life (USEPA 1986).

PERSPECTIVE

Although all uses of mirex as a pesticide have been canceled in the United States, its unique environmental persistence, along with its presence at the Salem, Ohio Superfund site, makes it necessary to evaluate its (1) potential adverse human health effects and (2) appropriate cleanup levels. Consequently, this report seeks to review the published scientific information on mirex, including its chemical, physical, and biological properties, and to summarize the environmental issues related to the presence of mirex at the Salem site so that risk managers can make appropriate remedial decisions.

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II. PHYSICOCHEMICAL PROPERTIES

GENERAL PHYSICAL AND CHEMICAL PROPERTIES

Mirex is a snow-white, odorless, free-flowing, synthetic, crystalline solid which is based on two linked five member rings. It has the molecular formula $C_{10}Cl_{12}$ and a molecular weight of 545.5. The Chemical Abstracts Service (CAS) 9th Collective Index preferred name for mirex is 1,1a,2,2,3,3a,4,5,5,5a,5b,6-dodecachlorooctahydro-1,3,4-metheno-1H-cyclobuta[cd]pentalene, and the CAS Registry Number is 2385-85-5. The International Union for Pure and Applied Chemistry (IUPAC) name for mirex is perchloropentacyclo-($5.2.1.0^{2,6}.0^{3,9}.0^{5,8}$)decane. Mirex has been known and marketed under many other names. In general, however, mirex refers to the pesticide or pesticide bait used to control imported fire ants while dechlorane refers to a fire-retardant additive used in a number of different products (Markin 1971; Waters 1976; WHO 1984; IARC 1979). Technical-grade preparations of mirex reportedly contain 95.19 percent mirex and 2.58 percent chlordecone (kepone) (NRC 1978).

The vapor pressure for mirex at 25° C is 3×10^{-7} mm Hg (IARC 1979). It is essentially insoluble in water but soluble in several organic solvents, including methyl ethyl ketone, carbon tetrachloride, benzene, xylene, and dioxane. The solubilities of mirex in a number of materials are listed below (Anonymous 1987; Hazard Handbook No.561; HSDB 1991; IARC 1979; NRC 1978; WHO 1984).

Water: < 1 ppb
Dimethyl sulfoxide: > 10 mg/ml at 24° C
Ethanol: 1-10 mg/ml at 24° C
Tetrahydrofuran: 381.4 mg
Carbon disulfide: 18%
Chloroform: 17%
Dioxane: 15.3%
Xylene: 14%
Benzene: 12%
Carbon tetrachloride: 7%
Methyl ethyl ketone: 5-6%

Mirex is extremely stable and does not react readily with sulfuric, nitric, hydrochloric, or other common acids; bases; chlorine; ozone; zinc dust; or sulfur trioxide (WHO 1984; Waters 1976). Mirex resists pyrolysis and has a melting point between 483° C and 487° C, with decomposition beginning at 525° C. Combustion is 98 to 99 percent complete at 700° C, yielding hexachlorobenzene, carbon monoxide, carbon dioxide, hydrogen chloride, chlorine, carbon tetrachloride, and phosgene vapor (WHO 1984).

Reductive dechlorination of mirex can be accomplished by reaction with reduced iron porphyrin or, more effectively, by reaction with vitamin B_{12} (Schrauzer and Katz 1978). Stojkovski et al. (1991) investigated the dechlorination of mirex using the Ni_2B in situ catalytic dechlorination technique. The catalytic reaction involved the substitution of hydrogen atoms for chlorine atoms, and produced compounds containing between 5 and 10 chlorine atoms.

FATE AND TRANSPORT

Slow, partial decomposition of mirex to photomirex (8-monohydromirex) by ultraviolet irradiation has been reported, and may be the fate of most mirex in the environment (WHO 1984). Photomirex has chemical and physical properties similar to mirex and kepone (Kaiser 1978). The half-life of mirex dispersed in water under intense UV radiation at 90° C to 95° C was reported by Knoevenagel and Himmelreich (1976) to be 48.4 hours. Several other photodegradation products have been reported to be found in the environment, including 10-monohydromirex; 5,10-dihydromirex; chlordecone (kepone); and 2,8-dihydromirex (WHO 1984, Carlson et al. 1976). A variety of soil microorganisms that have been tested failed to degrade mirex; however, Andrade and Wheeler (1975) reported that sewage sludge organisms, under anaerobic conditions, appear to degrade mirex to 10-monohydromirex and, possibly, to 9-monohydromirex.

Mirex is persistent in the environment due to its stability, and numerous examples of its persistence can be found in the literature. Carlson et al. (1976) reported that soil samples recovered 12 years after application of 1 part per million (ppm) mirex contained mirex and a number of mirex-related organochlorine compounds. The concentrations of mirex and its metabolites represented about 50 percent of the mirex applied. Kepone levels were approximately 0.02 ppm. In the same investigation, Carlson et al. reported on mirex ant bait which was recovered from the site of an airplane crash 5 years after the plane went down at the edge of a shallow pond. After 5 years, the organochlorine residues in the ant bait were 81.4 percent mirex and less than 2 percent kepone. Of the organochlorine residues in the pond muck samples, mirex was reported to make up 75.9 percent of the total and kepone was 5.7 percent.

Mirex has a very low vapor pressure (3.0×10^{-7} mm Hg at 25° C), and thus exists mainly in the particulate phase with a lesser amount in the vapor phase at ambient conditions (IARC 1979; Eisenreich et al. 1981). At locations where mirex dust may have been released into the atmosphere, such as near production facilities, aerial transport could have occurred (WHO 1984). The U.S. Environmental Protection Agency (USEPA 1978) estimated that people residing in areas where mirex-containing baits had been applied at rates of 1.7 to 3.4 g of

mirex per acre, may have inhaled 0.4-0.8 ng/day of mirex. Present-day incineration of plastics or other articles treated with dechlorane may also liberate mirex into the atmosphere.

Mirex has a low solubility in; concentrations of mirex above one μg/l will not dissolve in water, but will chemically adhere to particulate matter in the water (WHO 1984). Low levels of mirex (2-30 ng/l) were reported by Sandhu et al. (1978) in 12.5 percent of the wells tested in rural Chesterfield County in northern South Carolina. Mirex was detected in 72.7 percent of the wells in rural Hampton County in southern South Carolina. Recorded levels averaged 83 ng/l of potable water; the highest reported concentration was 437 ng/l. None of the more than 50 water samples from the Southwest contained mirex when tested at a detection level of 0.1 parts per billion (ppb). Any detectable mirex was associated with the suspended matter filtered from the water (USEPA 1978).

The logarithm of the mirex octanol water partition coefficient (K_{ow}) of 6.89 (Veith et al. 1979) indicates that mirex is likely to be strongly adsorbed by soil and soil organic matter, and is therefore likely to remain near the surface. Mirex residues in open-pasture soils after aerial mirex treatment have generally ranged from 0.1-10 ppb (MAC 1972). The major loss from terrestrial systems is likely to be through erosion and particle-bound runoff. Sediments act as a sink for mirex that has been leached and deposited in streams or ponds via runoff. Mud samples from ponds and drainage ditches have yielded mirex concentrations similar to those reported for soils (MAC 1972).

Although a Henry's Law constant for mirex of 5.16×10^{-4} atm-cu m/mole at 22° C (Yin and Hassett 1986) suggests that the volatilization half-life of mirex from a model river is 10.7 hours (Lyman et al. 1982), this estimate neglects the effect of adsorption of mirex to sediment and suspended matter. If these effects are considered, the half-life estimate is 1100 years (USEPA 1987).

ENVIRONMENTAL OCCURRENCE

Markin et al. (1971) reported that certain aquatic algae contain relatively high levels of mirex compared to the residues in the bottom sediments, indicating bioaccumulation of the compound.

Aquatic scavengers such as crayfish, shrimp, crabs, and snails, which consume sediment and other organic materials, also accumulate mirex. Bioconcentration factors of 12,200 for algae, 2,580 for fish, 4,900 for snails, 16,860-71,400 for crayfish, and 14,650 for daphnids have been reported. After 70 days of exposure to 0.038 μg/l of mirex in water, the following bioaccumulation factors were calculated: grass shrimp, 13,100-17,400; sheepshead

minnows, 28,900-50,000; mud crabs, 15,000-18,700; hermit crabs, 44,800-71,100; ribbed mussels (soft tissue), 42,000-52,600; and American oysters (soft tissue), 34,200-73,700 (Verschueren 1983). Huckins et al. (1982) exposed fathead minnows to three concentrations of [^{14}C]mirex for 56 days. Bioconcentration factors as high as 51,400 were reported, and the [^{14}C]mirex residue concentrations did not level off during the 56-day exposure period.

Borthwick et al. (1973) studied the movement and accumulation of mirex from treated lands and high marshes to estuarine biota near Charleston, South Carolina. All animal classes evaluated contained mirex, with higher biological concentrations occurring in predators (e.g., raccoons and birds). The residue ranges for the sample categories were: water, <0.01 ppm; sediments, 0-0.07 ppm; crabs, 0-0.60 ppm; finfish, 0-0.82 ppm; shrimp, 0-1.3 ppm; mammals, 0-4.4 ppm; and birds, 0-17.0 ppm. In a follow-up study (Borthwick et al. 1974), estuarine sediments, crabs, shrimp, and finfish were collected from 11 stations 2 years after aerial applications of mirex bait. No mirex was found in the bottom-sediment samples or in 36 animal samples. Mirex was present in three species of fish (white catfish, 0.021 ppm; bluegill, 0.047 ppm; and carp, 0.12 ppm) and in blue crabs (0.026 ppm) at two freshwater stations. This study indicates that mirex in crabs, shrimp, and finfish decreased in the 18 to 24 months between the two studies.

Collins et al. (1974) monitored the accumulation of mirex in 61 species from May 1971 to May 1972, following a single aerial application of mirex bait. The reported levels in most species were low (0.001-0.005 ppm), although certain birds did accumulate residues in the range of 1-8 ppm. The highest levels were seen in loggerhead shrikes and mockingbirds. Collins et al. (1974) attributed these levels to the diets of these birds. The birds with the greatest home range and those whose diets consisted mainly of seeds, fruits, and vegetables had lower mirex levels than the more carnivorous species, such as shrikes, mockingbirds, and meadowlarks.

The highest mirex level observed by Collins et al. (1974) in the four mammalian species sampled was 0.450 ppm in Eastern harvest mice, 1 month after treatment. Reptiles and amphibians accumulated mirex levels from 0.001-0.828 ppm. The range for mammals and birds was 0.001-8.483 ppm. Predatory or carnivorous fish, reptiles, and birds contained higher levels of mirex than the herbivorous or omnivorous species. The authors theorized that the high levels seen in birds, as compared to other carnivores, were related to the large volumes of food that birds consume per unit of body weight. Residues in most animals peaked 1-3 months after treatment and then declined.

Collins et al. (1974) also analyzed a number of items in the human food chain. Mirex was detected in 77 percent of the samples analyzed 1 year after treatment. Mirex was not detected in beef fat either before or 1 year after treatment. Low levels were detected in milk (0.001-0.022 ppm), chicken eggs (0.001-0.493 ppm), and chickens (0.004-0.515 ppm).

FDA conducted a survey in 1976 to measure mirex concentrations in fishery products from the seven Atlantic-Gulf states that had been treated with mirex to control fire ants. Data were obtained on 132 samples of finfish, shellfish, and crustaceans. None of the shellfish or crustaceans contained any detectable mirex, but some samples of finfish from all surveyed areas, except Texas, contained detectable mirex. Levels ranged from 0.002 ppm in Mississippi to 0.029 ppm in Alabama (USEPA 1978).

Terrestrial plants from mirex-treated areas have low (<0.05 ppm) residue levels. Markin et al. (1972) reported that bahiagrass contained 0.0003-0.017 ppm when grown in soils containing 0.001-0.002 ppm mirex. De La Cruz and Rajanna (1975) studied mirex uptake in soybeans, garden beans, sorghum, and wheat seedlings grown in soils containing 0.3-3.5 ppm mirex. The seedlings contained 0.01-1.71 ppm mirex, and observed levels were proportional to mirex levels in the soil.

Fish contaminated with mirex from mirex manufacturing facilities in the Lake Ontario area had mirex levels of <0.01-0.97 ppm. Nearly half of the species sampled contained mean levels of mirex above the Food and Drug Administration's (FDA) 0.1 ppm guideline for fish (Kaiser 1978). Widespread contamination of aquatic birds in the Great Lakes region was thought to be the result of mirex in Lake Ontario water and fish. Mirex has been found in the eggs of herring gulls, cormorants, gyrfalcons, peregrine falcons, prairie falcons, and pigeon hawks.

Studies involving continuous exposure to low levels of mirex have been reported, including exposures of chickens to low levels of mirex in feed (0.001-0.030 mg/kg) and in the soil of their pens (0.03-0.25 mg/kg). Resulting residues of mirex in the abdominal fat of the chickens were 0.072-1.09 mg/kg (Putnam et al. 1974). In a similar study, chickens were fed 0.01 mg/kg or 1.06 mg/kg mirex in the diet for 39 weeks. Residues in the tissues of the hens were 0.01-0.3 mg/kg and 0.3-25 mg/kg, respectively (Woodham et al. 1975).

Mirex has been reported in adipose tissues of humans. In one study, Kutz et al. (1974) detected 0.16-5.94 mg/kg mirex in fat samples of Georgia and Louisiana residents. Average residue levels in adipose tissues of residents from those Southern states where mirex was used against fire ants were 1.32 mg/kg (USEPA 1976). Traces of mirex have been found in human milk samples from women across Canada, with levels ranging from 0.1-0.6 μg/kg (wet weight basis) and 2.3-21.5 μg/kg (fat basis) (Mes et al. 1978). No advise health effects of mirex have been reported in any of the human environmental occurence studies.

Work by Mehendale et al. (1972) with rats showed that 58.5 percent of the orally administered dose of [^{14}C]mirex was excreted in the feces, and 0.69 percent was excreted in the urine after 7 days. Tissue storage was observed, with fat, muscle, liver, kidneys, and intestines containing 27.8, 3.20, 1.75, 0.76, and 0.23 percent of the total dose, respectively. The first half-life was 38 hours, but the second half-life was in excess of 100 days, indicating a slow rate of elimination from the body. No metabolites of mirex were detected in the feces, urine, or tissues examined.

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III. HEALTH EFFECTS IN HUMANS AND ANIMALS

INTRODUCTION

This section of the mirex report is modelled after the Health Effects chapters of the Toxicological Profiles compiled by the Agency for Toxic Substances and Disease Registry (ATSDR). It contains descriptions and evaluations of studies of mirex published in the scientific literature, and includes interpretations of data on the potential health effects associated with exposure to mirex. The governmental objective of the Health Effects chapters of the Toxicological Profiles is stated by the ASTDR as follows:

> Its purpose is to present levels of significant exposure for [mirex] based on toxicological studies, epidemiological investigations, and environmental exposure data. This information is presented to provide public health officials, physicians, toxicologists, and other interested individuals and groups with (1) an overall perspective of the toxicology of [mirex] and (2) a depiction of significant exposure levels associated with various adverse health effects.

In order to provide public health and regulatory officials with a background as to what is known about the potential adverse health effects from exposure to mirex, this section of the report has adopted the ATSDR format.

DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

ATSDR (1991) further clarifies the organization of the toxicological profile chapter on health effects with the following statements:

> To help public health professionals address the needs of persons living or working near hazardous waste sites, the data in this section are organized first by route of exposure -- inhalation, oral, and dermal -- and then by health effect -- death, systemic, immunological, neurological, developmental, reproductive, genotoxic, and carcinogenic effects. These data are discussed in terms of three exposure periods -- acute, intermediate, and chronic.

In addition to consideration of the route of exposure and possible type of health effects, ATSDR (1991) incorporates a statement in the toxicological profiles that explains the Agency's process for evaluating nonhuman toxicological studies and how the Agency evaluates the relative seriousness of the identified potential adverse health effects:

Levels of significant exposure for each exposure route and duration (for which data exist) are presented in tables and illustrated in figures. The points in the figures showing no-observed-adverse-effect levels (NOAELs) or lowest-observed-adverse-effect levels (LOAELs) reflect the actual doses (levels of exposure) used in the studies. LOAELs have been classified into "less serious" or "serious" effects. These distinctions are intended to help the users of the document identify the levels of exposure at which adverse health effects start to appear, determine whether or not the intensity of the effects varies with dose and/or duration, and place into perspective the possible significance of these effects to human health.

The significance of the exposure levels shown on the tables and figures may differ depending on the user's perspective. For example, physicians concerned with the identification of persons with the potential to develop such disease may be interested in levels of exposure associated with "serious" effects. Public health officials and project managers concerned with response actions at Superfund sites may want information on levels of exposure associated with more subtle effects in humans or animals (LOAEL) or exposure levels below which no adverse effects (NOAEL) have been observed. Estimates of levels posing minimal risk to humans (minimal risk levels, MRLs) are of interest to health professionals and citizens alike.

In order to fully consider the potential for mirex to cause adverse health effects in humans, the following sections, organized according to exposure routes, present a comprehensive survey of the published studies related to the toxicology of mirex.

Inhalation Exposure

Death

No poisoning cases or epidemiological reports were located in the scientific literature which permitted the evaluation of lethality in humans following inhalation exposure to mirex. No studies were located which permitted the evaluation of lethality in animals, other then birds, following inhalation exposure to mirex. In birds, an acute inhalation LD_{50} of 1400 ppm has been reported (Waters et al. 1977).

No studies were located which permitted the evaluation of the following effects in humans or animals after inhalation exposures to mirex.

Systemic Effects

Immunological Effects

Neurological Effects

Developmental Effects

Reproductive Effects

Genotoxic Effects

Cancer

Oral Exposure

Death

No poisoning case reports or epidemiology data were located which permitted evaluation of lethality in humans following oral exposure to mirex.

Acute Studies. In rats the oral LD_{50} of mirex administered in corn oil has been reported to range from 365 to 740 mg/kg of body weight with some variation between the sexes. When mirex was administered orally in peanut oil to male and female rats, the LD_{50} was found to be 3000 mg/kg (Gaines 1969; Gaines and Kimbrough 1970). In hamsters the single, oral, LD_{50} dose was 250 mg/kg in males and 125 mg/kg in females (Cabral et al. 1979). No deaths were observed in mongrel male dogs administered single doses of mirex orally at 125, 250, and 500 mg/kg. At three higher levels, five dogs per group were dosed at 1000, 1250, and 1500 mg/kg. No deaths occurred at the 1000 mg/kg level, and 3/5 of each of the two higher dose group animals died between days 3 and 11 after dosing. A "post mortem examination of the dogs did not reveal the presence of any gross pathological changes" (Larson et al. 1979).

Subchronic Studies. Pairs (16 males and 16 females per group) of prairie voles (Microtus ochrogaster) continuously fed a diet containing mirex at 0, 1, 5, 10, 15, or 25 ppm for 90 days exhibited progressive mortalities of 6, 9, 56, 69, 75, and 100 per cent, respectively, as reported by Shannon (1976) in a doctoral thesis. Deaths in the 5 to 25 ppm groups were significantly different from controls ($P<0.01$). Animals fed 5

ppm died within 34 to 106 days, whereas all the animals fed 25 ppm died after being on the mirex-containing diet for 32 to 78 days. The Shannon study, being a dissertation completed in partial fulfillment of the requirements for a doctoral degree in zoology, has not been subjected to the level of peer review usually given to experiments prior to publication in the scientific literature.

Ten Charles River rats per sex per test group were administered daily doses of 5, 20, 80, 320, or 1280 ppm of mirex in their diet for approximately 90 days. Mortality occurred only in the high-dose group (Larson et al. 1979).

Two purebred beagle dogs per sex per test group were administered daily doses of 4, 20, or 100 ppm of mirex in their diet for 13 weeks. Of the high dose groups, one female during week 10 and one male during week 13 died during the study. No other mortalities were seen (Larson et al. 1979).

Chronic Studies. Fifty-two F344/N rats per sex per test group were administered daily oral doses of 0.1, 1, 10, 25, or 50 ppm of mirex in their diet for 104 weeks. Mortalities of the 25 and 50 ppm groups of male rats were higher "($p<0.001$) as compared to those of the controls after week 86 and week 87, respectively." No significantly increased mortality rates were seen in male rats at 0.1 or 10 ppm of mirex in the diet as compared to the control group. No significant increase in mortality was seen between any groups of female rats in this study nor in a second study at dose levels of 50 and 100 ppm (NTP 1990).

Shannon (1976) continuously fed prairie voles mirex in the diet at 0.1, 0.5, 0.7, 1.0, and 5.0 ppm for a five month period, and found that only the 5.0 ppm animals exhibited a statistically significant mortality (70 per cent, $P < 0.01$).

Systemic Effects

Respiratory Effects. No studies were located which permitted the evaluation of respiratory effects in humans or animals following oral exposure to mirex.

Cardiovascular Effects. No studies were located which permitted the evaluation of cardiovascular effects in humans or animals following oral exposure to mirex.

Gastrointestinal Effects. No studies were located which permitted the evaluation of gastrointestinal effects in humans or animals following oral exposure to mirex.

Hematological Effects. No studies were located which permitted the evaluation of hematological effects in humans or animals following oral exposure to mirex.

Musculoskeletal Effects. No studies were located which permitted the evaluation of musculoskeletal effects in humans or animals following oral exposure to mirex.

Hepatic Effects. No studies were located which permitted the evaluation of hepatic effects in humans following oral exposure to mirex.

There are a number of published reports on the induction of potential adverse hepatic effects from orally administered mirex in rats, mice, dogs, and rabbits (WHO 1984). The following referenced reports briefly highlight some of the typical health-related results.

Liver hypertrophy was observed in male and female rats administered a diet containing 25 ppm (mg/kg) of mirex for 166 days (23.7 weeks) (Gaines and Kimbrough 1970). In another study, liver hypertrophy developed in 13 weeks in male rats receiving 80 ppm and higher of mirex in the diet and in females at a dose of 320 ppm (Larson et al. 1979). Liver enlargement was observed in female rabbits fed 20 ppm of mirex in their diet for 8 weeks (Warren et al. 1978), in male and female dogs fed 100 ppm of mirex in their diet for 13 weeks (Larson et al. 1979), and in male mice fed 30 ppm in the diet for 12 weeks (Pitz et al. 1979).

Liver cell changes were seen histological in male rats fed a diet containing as low as 1.0 ppm of mirex for 166 days. The changes included alteration in the architecture of the rough endoplasmic reticulum, increase in the numbers of free ribosomes, and proliferation of the smooth endoplasmic reticulum (Gaines and Kimbrough 1970). Induction of mixed-function enzymes was demonstrated in male rats administered mirex by gavage at levels as low as 1.0 mg/kg of body weight per day for 14 days (Villeneuve et al. 1977). A similar induction of liver enzymes was also produced in male rats fed 5 ppm of mirex in the diet for 13 weeks (Villeneuve et al. 1979), and at a level of 1 ppm in the diet for 14 days (Iverson 1976). Microsomal enzyme activity was induced in rabbits fed 20 ppm of mirex in the diet for 8 weeks (Warren et al. 1978).

Renal Effects. No studies were located which permitted the evaluation of renal effects in humans following oral exposure to mirex.

Adverse renal effects due to mirex were detected in male F344/N rats orally exposed to 10, 25, and 50 ppm in the diet for 104 weeks (NTP 1990). Some evidence of nephropathy was observed in both sexes, and in most of the control and dosed animals. However, in males the incidence of "marked severity" of nephropathy was significantly higher in the 3 higher-dose groups as compared to the control, 0.1-, and 1-ppm-dose groups. The incidence of nephropathy in the 3 higher-dose groups was 3 to 4 times higher than in the control group. Mild to moderate nephropathy was observed in all groups of females, and was not related to the dose of mirex. No other non-neoplastic lesions associated with mirex administration were evident.

Dermal/Ocular Effects. No studies were located which permitted the evaluation of dermal/ocular effects in humans or animals following oral exposure to mirex.

Immunological Effects

No studies were located which permitted the evaluation of immunological effects in humans as a result of oral exposure to mirex.

Two studies have been identified which assess the influence of mirex on antibody-mediated immunity in the chicken. In the earlier study, levels of immunoglobin M and G were depressed in chickens fed a diet containing 500 ppm (mg/kg) mirex for up to 5 weeks. However, no effects on antibody production were evident (Glick 1974). These findings are consistent with a second study in which chickens were fed diets containing mirex at 100 mg/kg for 40 days after hatching (Rao and Glick 1977).

Neurological Effects

No studies were located which permitted the evaluation of neurological effects in humans in relation to oral exposure to mirex.

However, a recent NTP technical report (1990) characterizes the symptoms and signs of mirex exposure as "gastrointestinal irritation with nausea, vomiting, diarrhea, malaise, headache, central nervous system excitation (including depression, and paresthesia, ataxia, confusion, convulsions, ventricular fibrillation, central nervous depression, and central nervous respiratory paralysis), (National Clearinghouse for Poison Control Centers, 1976)." The National Clearinghouse for Poison Control Centers was maintained by the U.S. Food and Drug Administration as a central resource for poison control centers. The April 1976 file card on mirex from the Clearinghouse lists The Pesticide Dictionary (1975) and "Manufacturer" as the sources of the information.

Efforts to further identify the primary references listed as the sources of the cited neurological effects of mirex exposure have been unsuccessful. It is possible that the listed effects actually reflect observed adverse human effects from ingestion of high levels of kepone, a pesticide chemically related to mirex, or of some other chlorinated pesticides.

Shannon's dissertation (1976) reports that prairie voles continuously fed mirex at 5 ppm and above exhibited signs of adverse nervous-system involvement (i.e., loss of balance, tremors, spastic gate, and incoordination). In an attempt to detect behavioral effects of long-term exposures to mirex, Shannon also evaluated twenty responses in first- and second-generation voles born to dams continuously fed mirex at 0.1, 0.5, and 1.0 ppm. Responses evaluated include the following: righting reflex, forelimb placing, hindlimb placing, postural flexion, postural extension, normal posture, forelimb-grasp-reflex, hindlimb-grasp-reflex, swimming, straight-line walking, rooting, vibrissae placing, visual placing, negative geotropism, bar-holding ability, cliff-drop aversion, eye opening, auditory startle, retrieval, and defense-of-young. The author claimed that "mirex exposure at levels of 0.1 and 0.5 ppm in the first-generation M. ochrogaster pups and maternal adults produced no apparent effects with regard to the following behavioral tests: righting reflex, forelimb placing, postural flexion and extension, normal posture, fore- and hindlimb-grasp-reflex, swimming, straight-line walking, rooting, visual placing, cliff-drop aversion, latency-to-retrieve and defense-of-young" (Table III-1). She reported that mirex exposure at 0.1 and 0.5 ppm in the first-generation pups did appear to delay maturation and increased the days to strong response in the hindlimb placing, negative geotaxis, and bar-holding ability tests. Similar results were reported by Shannon (1976) for the second-generation studies, with some variations, but no statistical evaluations were provided, and the results were too occasional and too varied to appear to be reflective of mirex-induced effects. Given the large number of observations made, the lack of any convincing responses or trends, and the behavioral/developmental inconsistencies of some of the reported observations, the Shannon study is of minimal value in determining neurological effects of mirex in the vole.

In a 1977 report, male rats were fed mirex in their diets at doses of up to 80 ppm for 8 weeks. Treatment-related effects of hyperactivity with attenuated startle response, increased emergence time, and decreased ambulation were reported (Reiter et al. 1977; WHO 1984). "Behavioral changes" were noted in rats intragastrically administered mirex at 5, 12.5, or 25 mg/kg of body weight (Dietz & McMillian 1979; WHO 1984).

In another neurological study, "adult male rats were fed diets containing mirex at 1.78 and 17.8 mg/kg of body weight for several weeks and were tested on a variety of

behavioral tasks. No differences in behavior were seen between control and treated animals." (Thorne et al. 1978; WHO 1984)

Developmental Effects

No studies were located which permitted the evaluation of developmental effects in humans in relation to oral exposure to mirex.

The developmental effects of oral exposures of experimental animals to mirex have been studied mainly in rats and mice with attention to embryotoxicity and the induction of cataracts, visceral anomalies, and adverse cardiac and vascular effects. Additionally, the Shannon (1976) study, mentioned previously (Oral Exposure, Neurological Effects), attempted to evaluate twenty responses in prairie-vole pups or dams fed 0.1 or 0.5 ppm mirex continuously for up to two generations, and failed to establish clear, biologically consistent evidence of mirex-related developmental effects under the conditions of the study. The significance of the Shannon study is severely limited because of insufficient detail and due to the failure of the author to statistically establish the reliability of the reported observations.

Groups of 15 male and 20 female weanling Sprague-Dawley rats were fed diets containing mirex at 5, 10, 20, or 40 ppm for 91 days prior to mating and throughout mating, gestation, and lactation periods. Litter sizes were decreased in all dosed groups. However, survival rates of pups were affected only in the high-dose group. Hepatic microsomal enzyme activities were increased in all dosed female and offspring groups. Analysis of fat and liver tissues for mirex residues showed a dose-related increase in adult females and a similar increase in pup livers. Hematological parameters were not affected, but histopathological effects in the livers and thyroids of mothers and pups were present in all dosed groups. A high incidence of over 50 per cent cataracts was found in female pups at all doses, but a similar incidence of cataracts was present only in the high-dose male pups (Chu et al. 1981).

In an early study of the developmental effects of mirex on mice, strains of BALB/C and CFW mice were fed mirex at a concentration of 5 mg/kg in their diet for 30 days prior to mating. Both strains showed a significant reduction in litter size and BALB/C mice showed an increase in parental mortality. Neither strain of mice fetuses was examined for congenital defects (Ware and Good 1967). An extensive inhibition of reproduction in mice was seen in animals fed mirex at a concentration of 17.8 mg/kg in their diet. Decreased reproduction was also seen by the same authors in groups of field mice fed mirex at a level of 1.8 mg/kg (Wolfe et al. 1979). Sherman rats fed mirex at a concentration of 25 mg/kg in the diet before and after mating had reduced litter sizes,

progenies with reduced survival rates, and cataract formation in surviving pups. Progenies from females maintained at mirex dose levels of 5 mg/kg diet produced normal neonates. Microscopic examinations of the livers of rats fed mirex at 25 ppm for 166 days showed an "increase in smooth endoplasmic reticulum and free ribosomes, myelin figures, [and] osmiophilic dense bodies." These microscopic lesions were not reported in the rats fed mirex at lower doses (Gaines and Kimbrough 1970).

The cataractogenic effect of mirex in mice was studied further in CD-1 mice and in Long-Evans and Sherman rats. Doses of 1 to 10 mg/kg/day were administered to rats and of 1.5 to 9 mg/kg/day to mice by gastric gavage to the maternal animals on postnatal days 1 through 4. Dose-related, statistically significant incidences of nonreversible cataracts developed in the neonate groups of both species. The lowest dose of mirex administered to either group did not produce this effect. The incidences of cataracts in the 10 mg/kg/day dosed Long-Evans and Sherman neonates were 53 and 59 per cent, respectively. In the 6 mg/kg/day dosed CD-1 mouse, the cataract neonate incidence was 14 per cent. Ophthalmoscopic examinations revealed "outlining" effects of the lens at higher incidences than that of the cataracts in the rat and mouse neonate groups. The available data also indicated that pups from dams dosed with mirex, but nursed by undosed mothers, developed a lower incidence of cataracts due to the lower level of neonatal exposure to mirex (Chernoff et al. 1976; Chernoff et al. 1979).

In another study, Wistar rats were gavaged daily with mirex at dosages of 0, 1.5, 3, 6, or 12.5 mg/kg body weight on days 6 through 15 of gestation. The high dosage produced "maternal toxicity, pregnancy failure, decreased fetal survival, reduced fetal weight and an increased incidence of visceral anomalies." Maternal effects and increased fetal incidence of visceral anomalies were observed at lower incidences at the 6 mg/kg dose but were minimal or absent at the two lower doses. The combined incidences of visceral anomalies for each of the 2 higher dose groups were statistically significant, and consisted of subcutaneous edema, scoliosis, cleft palate, short tail, and heart defects, primarily fleshy heart and enlarged atrium. There were no significant adverse effects on reproduction parameters in the three lower dosages (Khera et al. 1976).

Previous studies have reported fetal edema and prenatal death in offspring of rats administered mirex by gastric gavage (Chernoff et al. 1976; Khera et al. 1976). These results led to a study of the effects of prolonged mirex-induced swelling of the cardiovascular system of fetal and newborn rats. Pregnant Long-Evans rats were administered mirex in peanut oil by intragastric gavage at dosages of 5, 6, 7, or 10 mg/kg of body weight on days 8.5 to 15.5 of pregnancy. The fetuses were removed by laparotomies on day 18.5 of pregnancy, and were maintained attached to the uterus while electrocardiographs (ECGs) were made. The sites and extent of edema in each

fetus were also assessed. ECG's from 80 controls and 205 mirex-exposed fetuses were obtained. Abnormal heart rates and edema in mirex-exposed fetuses were dose-related and were greater than those of controls. The mean heart rate for control animals was 150 beats per minute, but ranged from 180 to 224 beats per minute in the mirex-exposed animals. Cardiac arrhythmias were proportional to the degree of edema. The incidence of first-degree heart block, as denoted by a prolonged PR interval in the ECG, was also dose-related and ranged from 20 per cent in the 5 mg/kg dose group to 77 per cent in the 10 mg/kg dose groups. Cardiac block and arrhythmia were dose-related, and were not present in the control fetuses (Grabowski and Payne 1980).

Pregnant Long-Evans rats were administered mirex in peanut oil by intragastric gavage at dosages of 0.1, 0.25, 0.50, or 1.0 mg/kg/day. The report states that the doses were administered during pregnancy at early organogenesis and at later maturational periods as well. However, cardiac data were presented mainly for the pups from the dams treated during the later periods of pregnancy, consisting of days 15.5 to 21.5. The surviving 5-day-old pups from 1.0 mg/kg/day dose dams had a 10 per cent incidence of arrhythmias, and 4 per cent "still had first-degree heart blocks." At a dose of 0.5 mg/kg/day, the only information presented was that "no pups were stillborn" and that 27 per cent of the pups died after birth. For the pups born of dams given mirex at 0.25 mg/kg/day, "the incidence of first-degree heart block at birth was 17 per cent." Five days later, these pups had an incidence of 35 per cent of cardiac arrhythmias, and an unreported incidence of "first-degree heart blocks." The report stated that "we are now testing a dose level of 0.1 mg/kg/day", and "only a few cardiac arrhythmias have been found in the pups of this series" (Grabowski 1983). The data presented do not permit a statistical analysis of the experimental results. The significance of this study and its report are limited since they contain only a minimum of data and insufficient information to support its conclusions and interpretations.

Reproductive Effects

No studies were located which permitted the evaluation of reproductive effects in humans in relation to oral exposure to mirex.

Several studies have been conducted on the reproductive effects of mirex in animals and have been described under the previous **Developmental Effects** section. These effects included decreased fertility and pregnancy failure, reduction in number and size of litter, decreased fetal weight, increased maternal and fetal mortalities, reduced survival of offspring, and reduced viability of neonates (Ware & Good 1967; Gaines and Kimbrough 1970; Khera et al. 1976; Chernoff et al. 1976; Wolfe et al. 1979; Grabowski and Payne 1980; Chu et al. 1981).

The prairie vole (M. ochrogaster) doctoral-dissertation study of Shannon (1976) was a multigenerational study of the reproductive performance and behavioral-development effects of mirex administered in the feed. The study had three main treatment groups with several dose groups within each study: single-generation, 90-day exposure (1, 5, 10, 15, and 25 ppm mirex); single-generation, continuous-exposure (0.1, 0.5, 0.7, 1, and 5 ppm mirex); and, multigeneration, continuous-exposure (0.1 and 0.5 ppm mirex). The ten reproductive parameters evaluated included adult mortality, percentage of mated pairs producing litters each generation, number of days from pairing of male and female to the birth of successive litters, number of days between consecutive litters, number of offspring per litter, number of litters produced per group, number of offspring per group, percentage of pups surviving to days 4 and 21, lactation index, and percent mortality of pups (Table III-2). Although the report claimed an effect of 0.1 ppm mirex on the percentage of pups surviving to 21 days, the effect only reached statistical significance in the third-generation litters, where survival was 84 per cent. Survivals for the first-, second-, and fourth-generation litters were 89, 84, and 100 per cent, respectively. The 100 per cent survival in the fourth generation litter, which had normal numbers of offspring per litter, numbers of litters per group, and numbers of offspring per group is a strong indication that 0.1 ppm mirex was actually a no-effect dose level. As noted previously, the significance of the Shannon study is severely limited because of insufficient detail and due to the failure of the author to statistically establish the reliability of the reported observations.

Genotoxic Effects

No studies were located which permitted the evaluation of genotoxic effects in humans in relation to oral exposure to mirex.

Only one study of an in vivo mutagenicity evaluation of mirex in animals was located; it consisted of a dominant lethal study. Male Wistar rats in groups of 20 were gavaged daily with mirex for 10 days at mirex dosages of 0, 1.5, 3.0, or 6.0 mg/kg of body weight. The dosed rats were mated with untreated females for 5 days. The pregnant rats were then killed 13 to 15 days after separation from the males, and the viable embryos, deciduomas, and *corpora lutea* were counted to assess the incidence of dominant lethal effects. This procedure was repeated with a different set of males and females every 5 days for a period of 70 days, each period being denoted as a mating trial (i.e., consisting of 14 mating trials). The numbers of viable embryos and deciduomas per pregnant female in the three mirex-dosed groups were not significantly different from those of the control group. The incidence of pregnancy was decreased only in the first trial mating group of the 6.0 mg/kg test animals, which mated 0 to 5

days after dosing. In all 13 groups mating periodically after each additional 5-day trial, there were no effects on the incidence of pregnancy at any dose level (Khera et al. 1976).

Mirex has been found to be mutagenically negative in reverse-mutation assays with Salmonella typhimurium and Escherichia coli with and without microsomal enzyme activation or with preincubation protocols and various Salmonella strains (NTP 1990).

Cancer

No studies were located which permitted the evaluation of the occurrence of cancer in humans in relation to oral exposure to mirex.

In animals, one study in mice and two studies in rats exist which permit the assessment of the development of cancer in animals chronically exposed to oral doses of mirex. In addition to these three oral studies, there exists a study of the development of cancer in animals given a single, subcutaneous injection of mirex.

In the mouse study, two hybrid strains of mice, (C57BL/6xC3H/Anf)F1 and the (C57BL/6xAKR)F1, received commercial-grade mirex (98 per cent pure). Each mouse, in groups of 18 males and 18 females of each strain, was administered daily gavage doses of 10 mg/kg of body weight in gelatin from 7 to 28 days of age. The mice were then placed on a diet containing 28 ppm of mirex, which, after 4 weeks, was changed to 26 ppm. All mice died before the scheduled sacrificed time of 18 months. Tumor incidences in the dosed mice (Table III-3) were statistically compared with untreated or gelatine-treated control groups. Hepatoma incidences were found to be statistically significant for each of the two sexes of the two strains ($P < 0.05$). The term hepatoma was applied to hepatic tumors "locally invasive often with massive involvement of the liver causing death." (Innes et al. 1969; NTIS 1968). The scientific value of the study was limited due to poor survival rates of test animals in the presence of high survival rates in controls, the use of a single test dose, the lack of clear histological differentiation of each "hepatoma," and limited information on "common other lesions" reported.

In a 1977 report of a carcinogenicity assay of mirex in Charles River CD rats (Ulland et al. 1977), 26 rats of each sex were fed 50 or 100 ppm of mirex in their diet for 18 months. However, during the first 10 weeks of the study the test animals received doses of only 40 and 80 ppm of mirex, respectively. The highest doses were estimated to be maximum-tolerated doses. Twenty untreated rats of each sex were used as untreated controls. All surviving animals were killed 24 months after the beginning of

the dosing period. Control rats had significantly higher survival rates than the mirex-dosed animals, which developed dose-related, life-shortening effects. Mortality effects in the high-dose rats began after about week 52, while no deaths occurred in controls until week 70 in females or week 78 in males. Of the mirex-dosed males, only 50 per cent of the animals survived to about week 85, and 50 per cent of the females survived to termination at week 104.

Hepatocellular neoplastic nodules and well-differentiated hepatic carcinomas were reported for both doses in both sexes. The relevance of neoplastic nodules to carcinomas is not known, but were not present in control animals and rarely occur spontaneously in rats. The incidences of these lesions (Table III-4) were statistically significant in the high-dose male rats. Eight nonhepatic tumors were also present in various-dosed animals. These consisted of 1 lipoma and 1 squamous-cell carcinoma of the ear-duct in low-dose males; 2 fibromas, 1 fibrosarcoma, and 1 squamous-cell carcinoma of the ear-duct in high-dose males; and 2 fibrosarcomas in high-dose females. These tumors were not found in controls. In addition, extensive treatment-related liver toxicity was reported "from fatty metamorphosis and megalocytosis of hepatocytes, cystic degeneration and necrosis and biliary hyperplasia with periportal fibrosis, to circumscribed areas of cellular alteration" (Ulland et al. 1977; NTP 1990).

In a recently reported toxicology and carcinogenicity study of mirex conducted in the late 1970s (NTP 1990), groups of 52 F344/N rats of each sex were fed diets containing 0, 0.1, 1.0, 10, 25, or 50 ppm of mirex for 104 weeks. Due to high survivals and the lack of observable toxic effects in female rats, a second study was conducted in which three similar groups of additional females were fed levels of 0, 50, and 100 ppm of mirex. The surviving animals were killed approximately 3 to 6 weeks after final dosings. During the study, there were no significant differences in survival rates between control groups and any of the dosed groups of female rats in either the first or second studies. In males, only the survival rates of the 25 and 50 ppm groups were significantly lower ($P < 0.001$) than that of the control group after week 86 and week 87, respectively.

In the male rat study and the two female-rat studies, there was a dose-related statistically significant increase in the number of rats with non-neoplastic liver lesions as compared to controls. These lesions consisted of fatty metamorphosis, hepatocytomegaly, necrosis, and angiectasis of the liver. Benign neoplastic nodules of the liver in male and female rats, hepatocellular carcinomas in male rats, and combined benign neoplastic nodules and hepatocellular carcinomas occurred in males with positive, mirex-exposure dose trends (Table III-5). The results of this study support the interpretation that liver carcinomas are not induced in rats at specific low levels of chronic exposures to mirex.

Additionally, the combined incidences of benign pheochromocytomas and malignant pheochromocytomas of the adrenal gland appeared to occur in positive, dose-related trends in male rats. In the 25 and 50 ppm male groups, these lesions were significantly increased as compared to controls. In females, the incidences of these combined lesions were of borderline statistical significance, and did not appear to be related to mirex.

Transitional cell papillomas of the kidneys in male rats developed in a positive, dose-related trend, but were of marginal statistical significance by the tumor-incidence test. The rarity of this neoplasia does not readily permit assessment of the potential of these papillomas to progress to carcinomas.

Mononuclear cell leukemia developed in a positive, dose-related trend in females in both the first and second studies. A low but significant increased incidence of 40 per cent in the 20 ppm dose was not supported by the incidence in the 50 ppm dose group, and the incidence in the control group was 31 per cent. The evidence of a mirex-related leukemia in male rats was equivocal.

The overall conclusion reached by the NTP Peer Review Group assessing the carcinogenicity test data for mirex was that there is "clear evidence" of mirex "carcinogenic activity for male and female F344/N rats" based on "increased incidences of benign neoplastic nodules of the liver, as well as by increased incidences" in males of benign "pheochromocytomas of the adrenal gland," an increased incidence of "transitional cell [benign] papillomas of the kidney in males and by increased incidences of mononuclear cell leukemia in females" (NTP 1990). The pathological classification of altered foci in the livers of rodents has changed since completion of the NTP review. Under current guidelines of the NTP, it is not clear how many of the benign neoplastic nodules in livers of treated rats would be classified as cancers. Consequently, calculation of a cancer slope factor for mirex on the basis of this study is not appropriate.

In another long-term observational study (NTIS 1968), 4-week-old mice were administered a subcutaneously injected dose of mirex once only and the surviving animals were killed after 18 months. Animals in groups of 18 of each sex and of each inbred strain [(C57BL/6xC3H/Anf)F1 and (C57BL/6xAKR)F1] were individually injected with mirex in 0.5 per cent gelatin at a dosage of 1000 mg/kg of body weight. Groups of 18 comparative animals were subcutaneously injected only with gelatin to be used as controls. More than 80 per cent of the dosed and control mice survived until the end of the study. At necropsy, the incidences of reticulum-cell sarcomas (lymphoid origin) in the two strains and sexes as well as the incidences of a similar combination of hepatomas in the dosed animals were significantly higher than those of the controls.

The incidences of these tumors as well as those of pulmonary adenomas are presented in Table III-6. The study indicates that a single-dose, subcutaneous, non-lethal exposure to mirex induced a significant neoplastic effect in mice within 18 months.

Dermal Exposure

Death

No poisoning cases or epidemiological study reports were located which permitted the evaluation of lethality in humans following dermal exposure to mirex.

Acute dermal LD_{50} values for rabbits of 800 mg/kg and of 2000 mg/kg in rats have been reported (Gaines 1969; Waters 1976). Additionally, a dermal toxicity study was located which permitted evaluation of lethality in animals following dermal exposure to mirex. A subchronic, percutaneous toxicity study in adult male and female rabbits was conducted by exposing the test animals to mirex containing ant-bait material. The bait material contained "15 per cent of a 1 per cent solution of mirex in soybean oil impregnated on corn cob grits." In two test-dose groups of 5 males and 5 females each, the depilated skin was abraded by scratching with a wire mesh in such a way as to penetrate the outer skin layer without producing any bleeding. In two other parallel test dose groups, the depilated skin was left intact. The abraded and intact sets of animals received a dosage of 3.33 or 6.67 g of mirex bait per kg of body weight. The control rabbit groups of males and females received 6.67 g of control bait per kg of body weight. The animals were exposed to the mirex bait or control bait 6 to 7 hours each day, 5 days per week, for 9 weeks. No mirex-related effects were seen "in body weights, hematologic values, or urinalysis findings." A slight skin erythremia and scaling was noted in the high-dose rabbits, but was not evident after 2 days without exposure. No signs "of intoxication during the exposure period" or signs of edema were present. No effects on organ weights were observed. No mirex-residue tissue analysis was reported. The data suggest that mirex, in bait preparations, is not "effectively" absorbed through the skin and produces only a low-level, local skin irritation (Larson et al. 1979).

No studies were located which permitted the evaluation of the following effects in humans or animals following dermal exposure to mirex.

Systemic Effects

Immunological Effects

Neurological Effects

Developmental Effects

Reproductive Effects

Genotoxic Effects

Cancer

TOXICOKINETICS

Absorption

Inhalation Exposure

No studies were located regarding the absorption of mirex in humans or animals following inhalation exposure.

Oral Exposure

No studies were located regarding the absorption of mirex in humans following oral exposure. Studies in animals indicate that absorption of orally administered mirex is not complete. Mehendale et al. (1972) administered mirex (1.5 or 6.0 mg/kg body weight) in corn oil by oral intubation to male rats (CD-1 strain; Charles River) and calculated that 55 per cent of the administered mirex was excreted in the feces within 48 hours. Consequently, the apparent absorption of the orally administered mirex was 45 per cent.

Dermal Exposure

No studies were located regarding the absorption of mirex in humans or animals following dermal exposure.

Distribution

Inhalation Exposure

No studies were located regarding distribution of mirex in humans or animals following inhalation exposure.

Oral Exposure

No studies were located regarding distribution of mirex in humans following oral exposure. Mehendale et al. (1972) evaluated the distribution of mirex in male rats 7 days after oral intubation of 6.0 mg/kg mirex in corn oil. With 34.19 per cent of the administered dose of mirex recovered in tissues and 59.19 per cent recovered in feces (58.5 per cent) and urine (0.69 per cent), the following tissues or organs had the indicated percentages of the originally administered mirex: fat, 27.8 per cent; muscle, 3.2; liver, 1.75; small intestine, 0.76; large intestine, 0.23; testes, 0.12; kidney, 0.09; lung, 0.08; brain, 0.07; stomach, 0.06; and heart, 0.03. Based on assumptions that the rats had 9 per cent body fat and 40 per cent muscular tissue, the authors estimated that 27.8 per cent of the administered mirex was stored in fat, and 3.2 per cent in muscle.

A computerized simulation of mirex pharmacokinetics in the rat following either oral or intravenous administration resulted in the generation of a three-compartment open system with parallel first-order elimination into the urine and feces (Byrd et al. 1982). The simulation of the intravenously administered mirex projected two peripheral compartments with mirex in the blood clearing into the rapidly equilibrating compartment and redistributing to a slowly equilibrating compartment over a period of several weeks. The slowly equilibrating compartment served as a mirex storage area.

Morgan et al. (1979) reported the relative concentrations of mirex in tissues of five Pitman-Moore minipigs fed mirex dissolved in corn oil and mixed into semisolid food for 7 successive days. Nine days after dosing, the concentrations of mirex in ppm in various tissues were as follows: backfat, 41.5; liver, 1.24; brain, 0.62; and kidney, 0.44.

Female rhesus monkeys given ^{14}C-mirex orally (one animal) or intravenously (two animals) at approximately 1 mg/kg body weight had similar tissue distributions of mirex (Wiener et al. 1976). Over the longest evaluation period -- 388 days -- very little of the mirex was excreted in the urine (< 0.2 per cent) and relatively little was excreted in the feces (7 per cent). After 388 days, approximately 86 per cent of the administered dose of mirex was still in the body fat; other sites of accumulation and retention

included the skin, adrenal gland, peripheral nerve, and thyroid. The authors noted that the mirex disappeared rapidly from the plasma and remained strongly bound in the fat with a low level of fecal and urinary excretion for the duration of the experiment.

Pittman et al. (1975) evaluated the pharmacokinetics of mirex distribution within female monkeys following oral or intravenous administration, and concluded that the model that best fit the observations was a mamillary, four-compartment open system which provided for the urinary excretion of mirex from a "central" compartment and the fecal excretion of mirex from a "fast" tissue compartment. The authors stated that the "model predicted that the accumulation of mirex into fat would be retarded by the presence of a 'slow' tissue compartment, so that distribution equilibrium would take about half a year."

Dermal Exposure

No studies were located regarding mirex distribution in humans or animals following dermal exposure.

Metabolism

No studies were located regarding mirex metabolism in humans. Several studies conducted to evaluate the potential metabolism of mirex in animals have been uniformly negative. Rats given a single oral dose of ^{14}C-mirex did not excrete any metabolites of mirex, nor was there any evidence of mirex metabolism in in vitro liver preparations from the rat, mouse, or rabbit (Mehendale et al. 1972). Another single-oral-administration study in rats also failed to identify any evidence of metabolism of mirex (Gibson et al. 1972), as did a study with repeated administrations of mirex to rats (Ivie et al. 1974).

Stein et al. (1976) and Stein and Pittman (1977) attempted to identify a metabolite of mirex found in monkey feces following intravenous administration of mirex. They concluded that low levels of either the 10-monohydro or 9-monohydro derivative were present, but postulated that the derivatives could have been formed by bacteria in the lower gut or feces.

No evidence of metabolism of mirex to kepone was found in minipigs fed mirex for 7 days (Morgan et al. 1979).

Excretion

Inhalation Exposure

No studies were located regarding the excretion of mirex in humans or animals following inhalation exposure.

Oral Exposure

No studies were located regarding the excretion of mirex in humans following oral exposure. In animals, several studies have been conducted to investigate the routes of and rates of mirex excretion following oral exposure. Mehendale et al. (1972) found that CD-1 rats fed mirex in corn oil by oral intubation excreted mirex primarily in the feces. The authors were unable to estimate the relative contribution of the fecal or urinary excretion routes to elimination of mirex because of the incomplete absorption of the orally administered mirex and the relatively short period of observation (7 days).

In a much longer study conducted with three female monkeys, Wiener et al. (1976) found that both orally and intravenously administered mirex were excreted in similar ways when evaluated after 21, 106, or 388 days. The total per cent of the original dose of mirex excreted via the feces or urine over 388 days was 6.91 and 0.18 per cent, respectively. The study had too few animals to establish a sense of the reliability of the observations, but the results were consistent between individual animals and gave an indication of the relative excretion of mirex by the two routes considered.

Dermal Exposure

No studies were located regarding the excretion of mirex in humans or animals following dermal exposure.

RELEVANCE TO PUBLIC HEALTH

No evidence has been found that acute, subchronic, or chronic exposures to mirex by the inhalation, oral, or dermal routes is toxic or carcinogenic to humans. Since occupational exposures to some chlorinated hydrocarbons have in the past produced the first recognizable toxic effects in workers, it is in occupational exposures that one would expect to first see

potential toxic effects of mirex on humans. A recent publication by NTP (1990) on mirex attributes a series of human symptoms to mirex exposures, including gastrointestinal irritation and varied central-nervous-system effects, including excitation, depression, and respiratory paralysis. The primary source of this information was cited by NTP as the "National Clearinghouse for Poison Control Centers (1976) Mirex." This information source is not available for verification, but it appears that the original information was not derived from reports of adverse human responses to exposures to mirex. Instead, the symptoms attributed to mirex appear to have been obtained, as noted previously, from a published study describing the adverse human effects of exposures to high levels of kepone at a manufacturing site, from studies of other chlorinated pesticides or from nonpublished information provided by the "manufacturer".

In that no adverse human health effects have been reported nor identified from any exposures to mirex, it is necessary to utilize information obtained from toxicological studies and experimental observations to assess what might be the potential adverse human health effects from exposures to low levels of mirex in the environment. The principal issue to be considered is the relevance of given levels of mirex in humans and in the environment to potential adverse health effects in exposed individuals. Several studies have indicated that the target organs and tissues of mirex toxicity in animals, but not reported in humans, are the liver and kidneys, as well as the immunologic, neurological, developmental, and reproductive systems. The results of the mirex studies discussed in the initial portion of this section are summarized in Table III-7. These mirex target sites were identified in studies of animals exposed primarily by the oral route. The toxic-effect data are relevant to public health in that mirex is widely distributed throughout the environment, primarily in trace amounts in sediment, soil, and food, and in human, animal, fish, and wildlife tissues. However, the lack of adverse health effects in persons occupationally exposed to mirex in the past, and the lack of any evidence of an association of adverse health in humans with chronic residue levels of mirex in their tissues, implies that the low levels currently present in the environment do not constitute a toxicological health problem.

Death

No evidence has been found about what mirex exposure levels, by any routes, would be potentially lethal to humans.

An acute inhalation LD_{50} toxicity in birds of 1400 ppm has been reported (Waters et al. 1977). In male and female rats, the oral LD_{50} ranged from 365 to 740 mg/kg in corn oil, and as high as 3000 mg/kg in peanut oil. The oral LD_{50} ranged from 125 to

250 mg/kg in hamsters, and was approximately 1000 mg/kg in dogs. Acute dermal LD_{50}s for rabbits of 800 mg/kg and of 2000 mg/kg in rats have been reported. Acute toxicities in domestic animals have not been reported.

In a subchronic toxicity study in which male and female rats were given daily diets containing mirex at 5 to 1280 ppm for 90 days, mortality occurred only in the highest dose groups. In male and female dogs fed 4, 20, or 100 ppm of mirex daily, only one male and one female died. In a chronic study, rats of each sex were fed mirex daily at doses of 0.1 to 50 ppm for 104 weeks. Increased mortalities occurred only in the 25 and 50 ppm male groups and not in the lower-three dose groups nor in any female groups. These findings indicate that subchronic exposures of animals or humans to environmental concentrations of mirex, which are much lower than the doses used in the subchronic studies, do not present a threat of mortality to humans.

Systemic Effects

No evidence has been found that exposure to mirex by any route produces any toxic systemic effects in humans.

Systemic effects involving mainly the liver have been studied in several species, including the rat, mouse, dog, and rabbit. Liver hypertrophy has been demonstrated in several subchronic studies involving mainly the rat and mouse, but also in the rabbit and dog. The lowest concentration producing this effect was in female rabbits fed 20 ppm in the diet for 8 weeks. Light and electron microscopic changes in liver cells were detected in male rats fed 1.0 ppm of mirex in their diet for 166 days. Liver microsomal enzymes were induced in male rats gavaged daily with mirex at 1 mg/kg of body weight for 14 days. This effect was also produced in rats at a level of 1 ppm in the diet for 14 days. Severe statistically significant nephropathy developed in male rats fed 10, 25, or 50 ppm of mirex in the diet for 104 weeks. However, the males at lower doses of 1 or 10 ppm in the diet were not affected.

Neurological Effects

No evidence has been found that exposure by any route to mirex produces toxic neurological effects in humans.

Behavioral changes in mirex-treated rats included hyperactivity, attenuated startle response, and decreased ambulation at a mirex dose of 80 ppm in the diet for 8 weeks. In another study, mirex was administered to rats by gavage 5 to 6 times per week 15

minutes before testing their behavior-response change rates to a pellet feeder. The rates of response were reduced by mirex and were often irreversible in nature at a dosage level of 5 mg/kg. In another behavioral study, adult rats fed levels of 17.8 mg/kg for "several" weeks showed no behavioral changes as compared to controls. Prairie voles fed a continuous diet supplemented with 5 ppm mirex exhibited neurological toxicities. Differences in behavioral response study results are commonly a consequence of test-methodology variations.

Developmental/Reproductive Effects

No evidence has been found that exposure to mirex by any route produces any toxic developmental or reproductive effects in humans.

The developmental and reproductive effects of mirex have been studied mainly in orally dosed rats, mice, and prairie voles, with particular emphasis on maternal and embryotoxicity and on the induction of cataracts, visceral anomalies, and adverse cardiac and vascular effects.

In pregnant rats and mice, oral administration of mirex has produced variable responses in increased maternal mortality and toxic effects at dose levels ranging from 1.8 to 40 ppm in the diet, with mice being more sensitive than rats. The toxic effects reported include: decreased litter size and pup survival, increased maternal and pup hepatic weights, histological changes in liver cells, alterations of microsomal enzyme activities, and increased histological changes in thyroid glands. Mirex dose-related, irreversible cataracts in rat and mouse neonates have been reported, with evidence that pups from mothers dosed with mirex but nursed by undosed dams developed lower incidences of cataracts. The effect was not seen in pups from mothers dosed at the lowest dosage tested of 1.5 mg/kg/day during pregnancy.

Prairie voles maintained on a continuous diet of feed supplemented with mirex at levels ranging from 0.5 to 25 ppm may have demonstrated various toxicities including adult mortalities at the highest doses to multigenerational effects on reproductive success at the lower levels. Animals fed mirex at 0.1 ppm did not exhibit clear evidence of adverse effects. As noted previously, the Shannon study (1976) is not of sufficient quality to use for setting regulatory standards.

Rats gavaged daily with mirex at dosages of 6 or 12.5 mg/kg of body weight during early gestation developed "maternal toxicity, pregnancy failure, decreased fetal survival, reduced fetal weight and an increased incidence of visceral anomalies." These effects were minimal or absent at dosages of both 1.5 or 3 mg/kg. There were no significant,

adverse reproductive effects in the three lower-dosage groups. Mirex administered to pregnant rats has been shown to produce dose-related cardiac edema, arrythmia, and first-degree heart block in fetuses and newborns. The effects were produced at a maternal dosage of 5 mg/kg of body weight, the lowest dosage tested. In a similar followup study using lower dosages, pups from dams receiving mirex at 0.25 mg/kg of body weight daily developed the three cardiac effects previously reported. Only a few cardiac arrhythmias were observed at the lower dose level of 0.1 mg/kg/day. The study contained limited information, and did not have sufficient data for statistical analysis.

Genotoxic Effects

No evidence has been found that exposure to mirex by any route produces genotoxic effects in humans.

The only in vivo study located of the potential genotoxic effects of mirex consisted of a dominant-lethal assay conducted in rats. Groups of male rats were gavaged daily for 10 days at three different dosage levels and were mated for 5 days with untreated females. Appropriate negative control groups were used. Fourteen mating trials of each dose group, including controls, were tested. Pregnant rats were killed 2 weeks after mating and viable embryos, deciduomas, and corpora lutea were counted. The pregnancy incidence was decreased in the first trial-mating group. This did not occur in any of the other trial-mating groups and the test parameters evaluated were not significantly different from the controls throughout the study. With mirex, all reverse mutation assays with Salmonella typhiurium and Escherichia coli, with and without microsomal enzyme activation or with preincubation protocols and various Salmonella strains, produced negative mutagenic findings.

Cancer

No evidence has been found that mirex exposure by any route produces cancer in humans.

Mirex has been evaluated for its carcinogenic potential in two strains of mice and two strains of rats using chronic, oral-administration protocols and in two strains of mice by a single-dose, subcutaneous protocol. In the studies using oral protocols, mirex has been reported to have produced statistically significant, benign, malignant, or combined (benign and malignant) liver tumors in mice and rats of both sexes. In the single-dose, subcutaneous-injection study in mice, mirex produced a statistically significant increase

in the incidence of both liver tumors and reticulum cell (lymphoid) sarcomas in both strains and both sexes of mice. The negative response of mirex to the dominant-lethal study in rats, and to all reverse mutation assays with and without microsomal enzyme activation or with preincubation test protocols, suggests an epigenetic mechanism of carcinogenesis, and that a threshold probably exists for its carcinogenic effects in animals. The IARC considers that "there is sufficient evidence" that mirex is carcinogenic in mice and rats.

LEVELS IN HUMAN TISSUE AND FLUIDS ASSOCIATED WITH EFFECTS

No information was located that associated mirex levels in human tissues and fluids with health effects.

LEVELS IN THE ENVIRONMENT ASSOCIATED WITH LEVELS IN HUMAN TISSUES AND/OR HEALTH EFFECTS

There are no quantitative data available that correlate environmental levels of mirex with adverse health effects in humans. However, numerous studies indicate that environmental exposures to mirex can result in detectable levels in human tissues (Kutz et al. 1979 and 1985, Greer et al. 1980, Williams et al. 1984, Frank et al. 1988, Burse 1989).

Analyses of lipid specimens throughout North America, especially in the southeastern U.S. where mirex was used as a pesticide and in the Great Lakes region where mirex was produced, reveal detectable levels of the chemical. In Louisiana and Georgia, a study was conducted to assess the levels of mirex absorbed by human adipose tissues in an intensely agricultural area where mirex was used for control of the imported fire ant. Fat samples obtained from postmortem examinations, and from specimens previously removed during therapeutic surgery, revealed mirex in the fat of 6 out of 1400 patients, with concentrations ranging from 160 ppb to 5940 ppb (Kutz et al. 1979). In a similar study, mirex was detected in 64 out of 624 lipid samples from the southeastern United States with the mean residue concentration being 286 ppb. The target populations of the study were residents during 1975 to 1976 of all counties in which mirex had been applied from 1965 to 1975 (Kutz et al. 1985). In a study designed to establish a partition coefficient for mirex in fat versus serum, the adipose tissues of residents living near a dump site in Memphis were obtained via needle biopsy. Nineteen out of 297 samples contained mirex. Concentrations were as high as 4680 ppb, with a mean concentration of 1080 ppb (Burse et al. 1989). In the northeast Louisiana area, fat samples removed at the time of surgery or taken within 24 hours after death were analyzed for the presence of various

organochlorine compounds. Mirex was found in 20 out of 22 samples, with a mean mirex residue concentration of 152 ppb and a range of 10 ppb to 600 ppb (Greer et al. 1980). In contrast, a follow-up study found mirex in only 2 out of 10 samples, at concentrations of 170 ppb and 120 ppb (Holt et al. 1986). These results indicate that mirex residue levels in humans evidently subside with time. In a study of residents of the Great Lakes region, human adipose tissue samples were obtained during autopsies. Mean concentrations from two cities were 27 ppb and 11 ppb, with the highest level detected being 190 ppb (Williams et al. 1984). In a study of Ontario residents exposed to mirex through industrial pollution, fat removed during autopsies was found to contain low levels of mirex in over 50 per cent of the samples (Frank et al. 1988).

Studies examining mirex levels in human serum have been conducted as well. In Tennessee, analyses revealed the presence of mirex in 13 out of 114 serum samples. The mean concentration level was 4.7 ppb, and residual levels ranged from 1.56 ppb to 16.8 ppb. These specimens were from a large cohort of persons living in close proximity to a dump site in Memphis (Burse et al. 1989). A study by Lloyd et al. (1974) in the southern United States revealed that 106 out of 142 serum samples tested contained mirex. The mean serum residue level was 500 ppb (Rouse et al. 1990). Several studies throughout the United States, and in Honduras as well, failed to detect any mirex in human blood (Murphy et al. 1983, Bush et al. 1984, Stehr-Green 1989, Steinberg 1989).

Human breast milk has also been analyzed for mirex. Mes et al. (1978) found mirex in 3 out of 14 samples of breast milk taken from women residing throughout Canada. Mirex has been found in Canada in fish from Lake Ontario, and there was some concern about mirex entering the human food chain. The 14 samples were selected based on observed gas chromatography patterns suggesting the presence of mirex in a previous human milk survey of polychlorinated biphenyls. Mirex levels ranged from 100 ppb to 600 ppb. Other studies in the U.S., Canada, and Finland did not detect mirex in milk (Barnett et al. 1979, Harrod and Asquith 1980, Savage et al. 1981, Bush et al. 1985, Mes et al. 1986, Mussalo-Rauhanaa et al. 1988).

It is evident from these studies that environmental exposures, either agricultural or industrial, can lead to detected levels of mirex in human tissues. Populations exposed to mirex in the 1970s in the rural South consistently showed elevated body burdens of mirex. However, none of these data indicate that environmental exposures to mirex causes adverse health effects in humans. In summary, although some quantitative data on levels of mirex in the environment and in various human tissues exist, there are no quantitative or qualitative data available that correlate environmental levels of mirex with adverse human health effects.

TABLE III-1
MIREX EFFECTS ON VOLE BEHAVIORAL PARAMETERS MULTIGENERATION, CONTINUOUS EXPOSURE STUDY

Parameter	First Generation		Second Generation	
ppm	0.1	0.5	0.1	0.5
1. Righting reflex	-	-	-	-
2. Forelimb	-	-	-	-
3. Hindlimb placing	+	+	-	-
4. Postural flexion	-	-	-	-
5. Postural extension	-	-	-	-
6. Normal posture	-	-	-	-
7. Forelimb-grasp-reflex	-	-	-	-
8. Hindlimb-grasp-reflex	-	-	-	-
9. Swimming	-	-	-	-
10. Straight-line walking	-	-	-	-
11. Rooting	-	-	-	-
12. Vibrissae placing	-	+	-	-
13. Visual placing	-	-	-	-
14. Negative geotropism	+	+	-	-
15. Bar-holding ability	+	+	-	-
16. Cliff-drop aversion	-	-	+	+
17. Eye opening	-	+	-	-
18. Auditory startle	-	+	-	-
19. Retrieval	-	-	-	-
20. Defense-of-young	-	-	-	-
Total	3/20	6/20	1/20	1/20

Source: Shannon, V.C. (1976)

TABLE III-2
MIREX EFFECTS ON VOLE REPRODUCTION PARAMETERS AS REPORTED BY SHANNON

Parameter	STUDY											
	Single Generation, 90-Day Exposure*					Single Generation, Continuous Exposure**					Multi-generation, Continuous Exposure ***	
ppm	1	5	10	15	25	0.1	0.5	0.7	1.0	5.0	0.1	0.5
1. Adult mortality	-	+	+	+	+	-	-	-	-	+	N/R	N/R
2. % Of mated producing litters each generation	+ (1,3,4)	+ (1,2,3)	+ (1,2)	+ (1,2)	N/A	-	-	+ (1,2,3)	+ (1,2,3)	+ (1,2,3)	-	-
3. Days from pairing to birth of litter	+ (1,2,3)	+ (1,2,3)	+ (1,2)	+ (2)	N/A	+ (3)	-	+ (3)	+ (3)	N/A	-	-
4. # Days between successive litters	-	-	-	+	N/A	-	-	-	-	N/A	-	-
5. # Offspring/litter	-	-	-	-	N/A	-	-	-	-	N/A	N/R	N/R
6. # Litters/group	+	+	+	+	N/A	-	+	+	+	+	N/R	N/R
7. # Offspring/group	+	+	+	+	N/A	-	-	+	+	+	-	-
8. % Pups surviving to: Day 4	-	-	-	-	N/A	-	-	+ (3)	+ (2,3)	N/A	-	+ (2)
Day 21	-	+ (1,2)	+ (1)	+ (1,2)	N/A	+ (3)	+ (3)	+ (3)	+ (3)	N/A	-	+ (2)
9. Lactation index	-	+ (1,2)	+ (1)	+ (1,2)	N/A	-	-	-	+ (3)	N/A	-	+ (1)
10. % Mortality of pups	-	+ (1,2)	+ (1)	+ (1,2)	N/A	+ N/R	+ N/R	+ N/R	+ N/R	N/A	-	+ (1,2)

KEY

( ) = generation showing positive findings

+ = reported to be a significant effect

- = negative

N/A = not applicable (no survivors)

N/R = not reported

* first generation only fed mirex in the diet for 90 days
** first generation only fed mirex in the diet for 5 months
*** all generations fed mirex in the diet for 5 months

Source: Shannon, V.C. (1976)

TABLE III-3

INCIDENCES OF HEPATOMAS IN TWO STRAINS OF MICE CHRONICALLY EXPOSED ORALLY TO MIREX

	CONTROLS		TREATED	
C57BL/6 HYBRID	**MALE**	**FEMALE**	**MALE**	**FEMALE**
C3H/Anf	8 of 79	0 of 87	6 of 18*	8 of 16*
AKR	5 of 90	1 of 82	5 of 15	10 of 16*

* $p < 0.05$

Source: Innes et al. 1969

TABLE III-4

INCIDENCE OF HEPATIC NEOPLASTIC LESIONS IN CD RATS CHRONICALLY EXPOSED TO MIREX IN DIET*

	NEOPLASTIC NODULES		HEPATOCELLULAR CARCINOMAS		COMBINED LESIONS	
	DOSE[a]					
SEX:	LOW	HIGH	LOW	HIGH	LOW	HIGH
Male	2/26	7/26	1/26	4/26	3/26	11/26**
Female	4/26	4/26	0/26	1/27	4/26	5/26

* No evidence of either neoplastic nodules or hepatocellular carcinomas in control groups made up of 20 males and 20 females, each.

** $P \leq 0.05$

[a] Dose levels were set at 40 and 80 ppm for the first 10 weeks of the study, after which they were increased to 50 and 100 ppm because only minor variations were observed between treated groups and untreated controls.

Source: Ulland et al. (1977)

TABLE III-5

INCIDENCE OF LIVER TUMORS IN RATS FED MIREX IN THE DIET FOR TWO YEARS

	ppm in diet						
	CONTROL	**0.1**	**1.0**	**10**	**25**	**50**	**100**
MALE							
Benign Neoplastic Nodules	3/52	5/52	5/52	14/52*	15/52*	26/52*	--
Hepatocellular Carcinomas	3/52	0/52	2/52	2/52	3/52	4/52	--
Combined Tumors**	6/52	5/52	6/52	15/52*	16/52*	28/52	--
FEMALE - First Study							
Benign Neoplastic Nodules	10/52	5/52	4/52	5/52	9/52	7/52	--
Hepatocellular Carcinomas	0/52	0/52	0/52	0/52	1/52	2/52	--
Combined Tumors**	10/52	5/52	4/52	5/52	10/52	9/52	--
FEMALE - Second Study							
Benign Neoplastic Nodules	2/52	--	--	--	--	23/52*	30/52*
Hepatocellular Carcinomas	0/52	--	--	--	--	0/52	1/52
Combined Tumors**	2/52	--	--	--	--	23/52*	31/52*

$P \leq 0.05$

** Represents total numbers of animals with either neoplastic nodules or carcinomas. If an animal displayed both types of tumors, only the hepatocellular carcinoma was scored.

Source: NTP (1990)

TABLE III-6

18-MONTH STUDY OF INCIDENCE OF NEOPLASTIC LESIONS IN TWO STRAINS OF MICE ADMINISTERED SINGLE SUBCUTANEOUS DOSES OF MIREX (1000 mg/kg)

STRAIN:	B6C3F1		B6AKF1		
SEX:	MALE	FEMALE	MALE	FEMALE	COMBINED*
Reticulum Cell Sarcoma					
Control	2/18	0/18	0/18	1/18	3/72
Test	6/18	0/17	1/17	3/18	10/70**
Hepatoma					
Control	1/18	0/18	1/18	0/18	2/72
Test	2/18	0/17	4/17	1/18	7/70**
Pulmonary Adenoma					
Control	1/18	0/18	0/18	0/18	1/72
Test	1/18	0/17	2/17	0/18	3/70

* Combined strains and sexes
** $P \leq 0.05$

Source: NTIS (1968)

TABLE III-7
LEVELS OF SIGNIFICANT EXPOSURE TO MIREX

FIGURE KEY	SPECIES	ROUTE	EXPOSURE FREQUENCY/ DURATION	EFFECT	NOAEL (mg/kg/day)	LOAEL (Effect) LESS SERIOUS (mg/kg/day)	LOAEL (Effect) SERIOUS (mg/kg/day)	REFERENCE
1	Dog/Mongrel	Gavage	1 exposure	Death	1000	—	1250	Larson et al. 1979
2	Rat	Diet	13 wk	Liver	.25	4	4	Larson et al. 1979
3	Dog/Beagle	Diet	13 wk	Liver	—	—	2.5	Larson et al. 1979
4	Rat	Diet	104 wk	Liver	0.05	—	0.5	NTP 1990
5	Rat	Diet	166 d	Liver	—	0.05	1.25	Gaines and Kimbrough 1970
6	Rabbit	Diet	8 wk	Liver	—	—	0.98	Warren et al. 1978
7	Mouse	Diet	12 wk	Liver	—	—	3.9	Pittz et al. 1979
8	Rat	Gavage	14 d	Liver	—	—	1.0	Villeneuve et al. 1977
9	Rat	Diet	13 wk	Liver	—	0.05	0.25	Villeneuve et al. 1979
10	Rat	Diet	14 d	Liver	0.0125	0.05	—	Iverson 1976

TABLE III-7 (continued)
LEVELS OF SIGNIFICANT EXPOSURE TO MIREX

FIGURE KEY	SPECIES	EXPOSURE ROUTE	EXPOSURE FREQUENCY/ DURATION	EFFECT	NOAEL (mg/kg/day)	LOAEL (Effect) LESS SERIOUS (mg/kg/day)	LOAEL (Effect) SERIOUS (mg/kg/day)	REFERENCE
11	Rat/F344/N	—	—	Kidney	—	—	0.5	NTP 1990
12	Chicken	Diet	5 wk	Immune system	20	25	—	Glick 1974
13	Chicken	—	40 d	Immune system	—	—	5	Rao and Glick 1974
14	Rat	Diet	8 wk	Neurologic	—	—	4	Reiter 1977 WHO 1984
15	Rat	Gavage	5-6 d/wk	Neurologic	—	—	5	Dietz and McMillan 1979 WHO 1984
16	Rat	Diet	"Several weeks"	Neurologic	0.00178	—	—	Thorne et al. 1978 WHO 1984
17	Rat	Diet	106 d through gestation/lactation	Reprod/ Develop	—	—	0.25	Chu et al. 1981
18	Mouse	Diet	30 d	Reprod/ Develop	—	—	0.65	Ware and Goode 1967

TABLE III-7 (continued)
LEVELS OF SIGNIFICANT EXPOSURE TO MIREX

FIGURE KEY	SPECIES	ROUTE	EXPOSURE FREQUENCY/ DURATION	EFFECT	NOAEL (mg/kg/day)	LOAEL (Effect) LESS SERIOUS (mg/kg/day)	LOAEL (Effect) SERIOUS (mg/kg/day)	REFERENCE
19	Mouse	Diet	15 mo	Reprod/ Develop	—	0.23	2.3	Wolfe et al. 1979
20	Rat	—	166 d	Reprod/ Develop	—	.05	1.25	Gaines and Kimbrough 1970
21	Rat	Gavage	1-10 mg/kg/d	Reprod/ Develop	—	—	10	Chernoff 1976
22	Mouse	Gavage	~ 3 wk	Reprod/ Develop	—	—	6	Chernoff 1979
23	Rat	Diet	Gestation 6-15 d	Reprod/ Develop	3	—	6	Khera 1976
24	Rat	—	Gestation 8-15 d	Reprod/ Develop	—	—	5	Grabowski and Payne 1980
25	Rat	Diet	Gestation 15.5-21.5 d	Reprod/ Develop	—	1.5	6	Grabowski 1983
26	Rat	Gavage	Gestation 6-15 d	Genotoxicity	6.0	—	—	Khera et al. 1976

TABLE III-7 (continued)
LEVELS OF SIGNIFICANT EXPOSURE TO MIREX

FIGURE KEY	SPECIES	ROUTE	EXPOSURE FREQUENCY/ DURATION	EFFECT	NOAEL (mg/kg/day)	LOAEL (Effect) LESS SERIOUS (mg/kg/day)	LOAEL (Effect) SERIOUS (mg/kg/day)	REFERENCE
27	Mouse	Gavage	≤ 18 mo	Hepatomas	—	—	3.4	Innes et al. 1969
28	Rat	Diet	18 mo	Tumors	2.5	—		Ulland et al. 1977
29	Rabbit	Dermal	6-7 hr/d, 5 d/wk 9 wk	Irritation/ edema	3.33	6.67	—	Larson et al. 1979

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IV. CURRENT REGULATORY STATUS OF MIREX

USEPA/SAFE DRINKING WATER ACT

The Safe Drinking Water Act requires the U.S. Environmental Protection Agency (USEPA) to establish maximum contaminant level goals (MCLGs) and national primary drinking water regulations (NPDWRs) for contaminants that may have adverse effect on human health and that are known or are anticipated to occur in public water systems. The MCLGs are non-enforceable health goals and the MCLs are enforceable standards. The MCLs must be set as close to the MCLG as is feasible with the use of the best technology, treatment techniques, and other means that are available. The Safe Drinking Water Act also provides for setting the national secondary drinking water regulations (NSDWRs) to control water color, odor, appearance, and other characteristics affecting consumer acceptance of water. The NSDWRs are not federally enforceable but are considered guidelines for the United States. There currently are no proposed or final goals, standards, or guidelines for exposure to mirex in drinking water under the Safe Drinking Water Act. The Ohio EPA also has not established a mirex water-quality standard for public water supplies.

USEPA/CLEAN WATER ACT

The Clean Water Act requires the USEPA to promulgate and to periodically update ambient-water-quality criteria. These criteria are to accurately reflect the latest scientific knowledge on (1) the kind and extent of all identifiable effects on health and welfare; (2) the concentrations and dispersals of pollutants, or their byproducts, through biological, physical, and chemical processes; and (3) the effects of pollutants on biological community diversity, productivity, and stability. These criteria are not rules and they do not have regulatory impact. Rather, they present scientific data and guidance on the environmental effects of pollutants that the USEPA considers to be useful in deriving regulatory requirements based on consideration of water quality impacts. The USEPA has recommended an ambient-water-quality criterion for mirex of 0.001 μg/l for both freshwater and marine aquatic life (USEPA 1986). The criteria for mirex were promulgated in 1976, and have not subsequently been revised. The Ohio EPA Water Quality Standard for Aquatic Life Habitat (a standard similar in intent to the USEPA ambient-water-quality criteria) is also 0.001 μg/l, expressed as a 30-day average.

U.S. DEPARTMENT OF AGRICULTURE

The U.S. Department of Agriculture (USDA) currently uses an action level of 0.1 ppm in its Food Safety and Inspection (FSIS) residue-monitoring programs for mirex in the fat of meat

and meat byproducts of livestock, including cattle, goats, swine, horses, sheep, poultry, and rabbits (51 FR 45114).

U.S. FOOD AND DRUG ADMINISTRATION

The Food and Drug Administration (FDA) has established an action level of 0.1 ppm for mirex in the edible portions of fish (52 FR 11549).

USEPA HEALTH EFFECTS

The USEPA, in its Health Effects Assessment Summary Tables (HEAST, USEPA 1991), reports identical subchronic and chronic oral reference doses (RfDs) of 2×10^{-6} mg/kg/day for mirex. Only the chronic oral RfD for mirex has been verified in the USEPA's Integrated Risk Information System (IRIS 1991) by the RfD/RfC Work Group. The verification date for mirex is given as April 15, 1987, and the review dates listed for this compound in the IRIS are June 24, 1986, and April 15, 1987. According to the HEAST (USEPA 1991), the USEPA has not determined subchronic or chronic inhalation RfCs for mirex.

The USEPA, in its HEAST (USEPA 1991), identifies mirex as a Group B2 carcinogen for exposure by inhalation or ingestion. This designation characterizes mirex as a probable human carcinogen based on sufficient evidence in experimental animals, but inadequate data in humans. A cancer slope factor for inhalation exposure to mirex has not been determined by the USEPA. However, an oral slope factor of 1.8 $(mg/kg/day)^{-1}$ has been reported for mirex in the HEAST (USEPA 1991). According to the HEAST, this value is based on the occurrence of liver and adrenal tumors in a 2-year dietary study in rats (NTP 1987). This value has not been verified by EPA's Carcinogen Risk Assessment Verification Endeavor (CRAVE) work group, and has not been reported in IRIS (1991). The derivation of the slope factor for oral exposure to mirex is described in USEPA's Health Effects Assessment for Mirex (USEPA 1987).

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V. REVIEW OF EXISTING HAZARD CRITERIA AND STANDARDS

Two primary types of criteria or standards are important in developing alternatives for remediation of the former Nease Chemical Corporation site: (1) criteria intended to protect the environment and biota, and (2) criteria intended to protect human health. Criteria likely to be considered by federal, state, and local agencies as a basis for deriving cleanup goals in any remedial alternatives are addressed in this section. The derivation of these criteria is briefly reviewed.

ENVIRONMENTAL CRITERIA AND STANDARDS

In general, there are few categories of individual chemical criteria that are directly intended for protection of the environment and environmental receptors. For mirex, only water-quality criteria for the protection of aquatic life are currently available. These are the most common types of chemical criteria that have been derived by regulatory authorities. Air, soil, and sediment criteria for protection of environmental receptors are, in general, less available. Typically, the criteria for these media, if needed, must be developed on a case-specific basis.

Under the Clean Water Act, the U.S. Environmental Protection Agency (USEPA) has recommended ambient water quality criteria of 0.001 μg/l for both freshwater and marine aquatic life (USEPA 1986). These criteria were promulgated in 1976 and have not been subsequently revised. The values do not have regulatory impact; rather, they present scientific data and guidance on the environmental effects of pollutants, and the USEPA considers them to be useful in deriving regulatory requirements. The freshwater criterion was based on the observation of effects in crustaceans (crayfish) at 0.1 μg/l with an application factor (i.e., uncertainty factor) of 0.01. Similar results were observed in a variety of marine crustaceans, although only after longer exposure times. The Ohio EPA Water Quality Standard for Aquatic Life Habitat (a standard similar in intent to the USEPA ambient-water-quality criteria) is 0.001 μg/l as well, expressed as a 30-day average.

HUMAN HEALTH CRITERIA

In this section criteria and standards applicable to residues in food products and exposure levels in humans are briefly discussed. The criteria for human exposure levels involve protection against both potential noncarcinogenic systemic effects and potential cancer effects.

Action Levels For Residues In Food Products

The U.S. Department of Agriculture (USDA) currently uses an action level of 0.1 ppm in its Food Safety and Inspection (FSIS) residue-monitoring programs for mirex in the fat of meat and meat byproducts of livestock, including cattle, goats, swine, horses, sheep, poultry, and rabbits (51 FR 45114). The Food and Drug Administration (FDA) uses an action level of 0.1 ppm for mirex in the edible portions of fish (52 FR 11549). Background documentation on development of the criteria for mirex in food products is being assembled under a Freedom of Information Act request. It appears that, at least for fish, the criterion was probably derived by analogy with kepone. Furthermore, it is likely that economic factors were also important considerations in deriving this action level.

Slope Factor

The slope factor is the slope of the dose-response curve in the low-dose region for an agent considered to be a potential carcinogen. It is an expression of the cancer risk (proportion affected) per unit of dose (typically expressed in mg/kg/day). Slope factors are sometimes expressed in units of risk per concentration unit of the agent in a given medium (e.g., air, water, food). To estimate the cancer risk above the normal background risk (i.e., excess risk) that is associated with a potential carcinogen, the slope factor is multiplied by the appropriate dose or concentration measurement.

Mirex is listed as a weight of evidence Group B2 carcinogen for exposure by inhalation or ingestion in the USEPA Health Effects Assessment Summary Tables (HEAST, USEPA 1991). This designation characterizes mirex as a probable human carcinogen based on the Agency's determination that sufficient evidence exists in experimental animals, although there is inadequate evidence in humans. A cancer slope factor for inhalation exposure to mirex has not been determined by the USEPA. However, an oral-exposure slope factor of 1.8 $(mg/kg/day)^{-1}$ has been reported for this compound in the HEAST. This value has not been verified by USEPA's Carcinogen Risk Assessment Verification Endeavor (CRAVE) work group and has not been reported in USEPA's Integrated Risk Information System (IRIS

1991). The documentation for USEPA's weight of evidence characterization and slope-factor derivation for mirex is presented in the Health Effects Assessment (HEA) for Mirex (USEPA 1987).

The only epidemiological study on cancer incidence related to mirex exposure appears to be an investigation of residents of the Love Canal area in New York. There is no evidence that elevated cancer rates are associated with residence in this area (Janerich et al. 1981, USEPA 1987). In the HEA USEPA (1987) considered in detail three animal studies on the carcinogenicity of mirex (Innes et al. 1969, Ulland et al. 1977, NTP 1987), and concluded that there was sufficient evidence that mirex is carcinogenic in experimental animals. Data for calculating the mirex cancer slope factor were based on the National Toxicology Program's carcinogenicity bioassay in rats exposed to mirex in the diet (NTP 1987).

As reported in the HEA (USEPA 1987), pooled counts were developed using the individual animal data appended to the NTP (1987) report. For male rats, the counts included any animal having a liver neoplastic nodule, hepatocellular carcinoma, adrenal pheochromocytoma, or malignant pheochromocytoma. For female rats in each of two studies, the counts included any animal having a hepatic neoplastic nodule, hepatocellular carcinoma, or leukemia. The resulting slope factors were 1.8, 0.8, and 1.2 $(mg/kg/day)^{-1}$ for the males, first-study females, and second-study females, respectively. The estimate of 1.8 $(mg/kg/day)^{-1}$ was chosen to represent a conservative estimate of the cancer potency for mirex.

It is unclear what factors were considered in evaluating the slope factor that was ultimately selected for mirex. For example, the final version (NTP 1990) of the 1987 draft NTP bioassay was not used by the USEPA in the slope-factor derivation presented in the HEA USEPA (1987), and it has not been determined if any significant changes subsequently occurred in the report. In addition, the present slope-factor estimate includes benign tumor counts for two endpoints (liver and adrenal tissue) based on their presumed potential to progress to malignant tumors. It is not clear if examination of the original bioassay slides using current guidelines for classification of tumors would significantly alter conclusions drawn from the bioassay studies, or change the value of the cancer slope factor. Finally, consideration of alternative dose-response modeling approaches for mirex (e.g., using pharmacokinetic or mechanism-of-action data) could also result in different estimates of the potency of this compound.

Reference Dose (RfD) For Noncarcinogenic Effects

A reference dose (RfD) is an estimate, developed by the USEPA and verified by the intra-agency RfD work group, with uncertainty spanning up to an order of magnitude, which

approximates the daily exposure that the human population, including sensitive subgroups, can tolerate over a lifetime without appreciable risk of deleterious effects. The RfD is a benchmark dose derived from the no-observed-adverse-effect-level (NOAEL) via the consistent application of order-of-magnitude uncertainty factors that reflect the quality of the data analyzed. In addition, a modifying factor is sometimes used that is based on a professional evaluation of the entire data base for an agent. USEPA guidelines (e.g., IRIS 1991) describe the use of uncertainty and modifying factors in deriving RfDs as follows:

- Use a 10-fold uncertainty factor when extrapolating from valid experimental results in studies using prolonged exposure to average healthy humans to account for the variation in sensitivity among members of the human population.

- Use an additional 10-fold uncertainty factor when extrapolating from the valid results of long-term studies on experimental animals when results of studies of human exposure are not available or are inadequate. This factor is intended to account for the uncertainty involved in extrapolating from animal data to humans.

- Use an additional 10-fold uncertainty factor when extrapolating from less than chronic results in experimental animals when there are no useful long-term human data.

- Use an additional 10-fold uncertainty factor when deriving an RfD from a lowest-observed-adverse-effect-level (LOAEL), instead of a NOAEL.

- Use professional judgement to determine the modifying factor, which is an uncertainty factor greater than zero and less than or equal to 10. The magnitude of the modifying factor depends on the professional assessment of scientific uncertainties of the study and database not explicitly treated by the other uncertainty factors, e.g., the completeness of the overall database or the number of species treated. The default value for the modifying factor is 1.

The RfD is determined by dividing the NOAEL by any uncertainty or modifying factors that are found to be appropriate, and is generally expressed in units of mg/kg/day. It is considered by the USEPA to be useful as a reference point from which to gauge the potential effects of the chemical at other doses. Usually, doses lower than the RfD are not associated with adverse health risks and are less likely to be of regulatory concern. As the frequency or the magnitude of the exposures exceeding the RfD increase, the probability of adverse effects in some members of a human population increases. However, it should not be categorically

concluded that all doses below the RfD are "acceptable" (i.e., risk free), and that all doses in excess of the RfD are "unacceptable" (i.e., resulting in adverse effects).

The USEPA, in its HEAST (USEPA 1991), reports identical subchronic and chronic oral RfDs of 2×10^{-6} mg/kg/day for mirex. However, only the chronic oral RfD for this compound has been verified in the USEPA's IRIS (USEPA 1991) by the RfD work group. According to IRIS, the USEPA has not determined subchronic or chronic reference concentrations (RfCs) for inhalation exposures to mirex.

The RfD for mirex is based on a multigenerational, continuous exposure study in which prairie voles were exposed to mirex in food at dietary levels of 0, 0.1, or 0.5 ppm (Shannon 1976). Reportedly, at 0.1 ppm, there was a statistically significant decrease in the survival of pups at 21 days and an increase in pup mortality. An uncertainty factor of 100 was used to account for intra- and interspecies differences. An additional factor of 100 was used because a no-observed-effect-level (NOEL) was not reached, the study was not of chronic duration, and the data base was insufficient to determine the most sensitive toxicologic endpoint.

The USEPA's overall confidence rating for the oral RfD is low. IRIS (1991) notes that the principal study appears to be of fair quality, but that a NOEL was not reached; thus, a low to medium confidence rating was assigned. In general, because the data base on chronic toxicity is considered to be incomplete and consists of studies that are not core graded, the oral RfD is given a low confidence rating.

In March 1989 (54 FR 9386), the USEPA proposed Guidelines for Developmental Toxicity Risk Assessment. The proposal was referred to as Proposed Amendments to the Guidelines for Health Assessment of Suspect Developmental Toxicants and were promulgated by the EPA in September 1986 (51 FR 34028). In the Proposed Guidelines, USEPA introduced the term RfD_{DT} for the reference dose for developmental toxicity derived from dividing the NOAEL for developmental toxicity by an uncertainty factor. This designation was intended to demarcate the developmental toxicity reference dose, which is based on a short-term exposure as occurs in most developmental toxicity studies, from the RfD, which the USEPA derives based on chronic or (sometimes) subchronic exposure scenarios.

The Proposed Guidelines note that uncertainty factors for developmental toxicity generally include a 10-fold factor for interspecies variation and a 10-fold factor for intraspecies variation. Additional factors also may be applied due to a variety of uncertainties that exist in the data base. Where only a LOAEL is available, questions relative to the sensitivity of end points reported, adequacy of dose levels tested, or confidence in the LOAEL reported, may require the use of an additional uncertainty factor of 10. The EPA does make the point, however, that, in general, an additional uncertainty factor is not applied for duration of

exposure. This is inconsistent with the approach used in developing the IRIS oral RfD for mirex. In that case, an additional uncertainty factor of 10 appears to have been incorporated to derive an RfD for chronic exposure to mirex, even though other experimental studies dealing with toxicity endpoints other than developmental effects were available.

It is clear that the Shannon (1976) reproduction study of prairie voles is not the best study to use as a basis for a chronic RfD for mirex. An evaluation of the statistical procedures used to analyze data in this study suggest that inappropriate statistical techniques were used, and that an overly large number of parameters were evaluated for statistical differences from control values. Some data appear to be incompletely reported. A preliminary reanalysis of the critical effects using more appropriate statistical techniques indicates that the decreases in pup survival and the increases in mortality were not significant at the 0.1 ppm dose level. Thus, the 0.5 ppm dose is a more accurate representation of the LOAEL. In addition, it is not clear that incorporation of an additional safety factor of 10 to account for chronic exposure is appropriate since the effects under consideration (developmental effects) take place over less-than-chronic time periods. It may be helpful to consider the relative persistence of mirex in biological tissues when selecting uncertainty factors. However, this approach does not appear to have been adequately described or documented.

It may be appropriate to reevaluate the basis for the current RfD for oral mirex in order to establish a critical endpoint and LOAEL or NOAEL. The selection of a NOEL of 0.1 ppm versus a LOAEL of 0.5 ppm could result in an increase of the existing value by a factor of 5. Detailed evaluation of the appropriateness of the uncertainty factors used could result in a more accurate estimate of potential exposure. It should be noted that the use of a total uncertainty factor of 10,000 is unusual in the IRIS database. Another outcome of such an analysis could be the selection of a different critical study from among those summarized in the Health Effects section of this report. Preliminary evaluation of these alternatives indicate that it is unlikely that a lower RfD would result if the Shannon (1976) study was determined to be inadequate.

Other approaches have been suggested for deriving allowable daily intakes for exposure to potentially toxic substances. These approaches have often focussed on developing ways to consider more of the available data when drawing the dose-response curve for a particular substance and when deciding on the appropriate safety factors to use. Dourson (1986) and others have described a graphic method of using available toxicologic data in developing guidelines for exposure to potentially toxic materials. For example, the data in Table V-1 and Figure V-1 show how more of the available toxicological data could be used to show observed toxic effects for different exposure times. Information developed in this manner could be used to generate a dose-response curve, perhaps with an estimated upper bound on response, to derive a protective RfD, or to demonstrate the large margin of safety associated with the existing RfD.

TABLE V-1
LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO MIREX

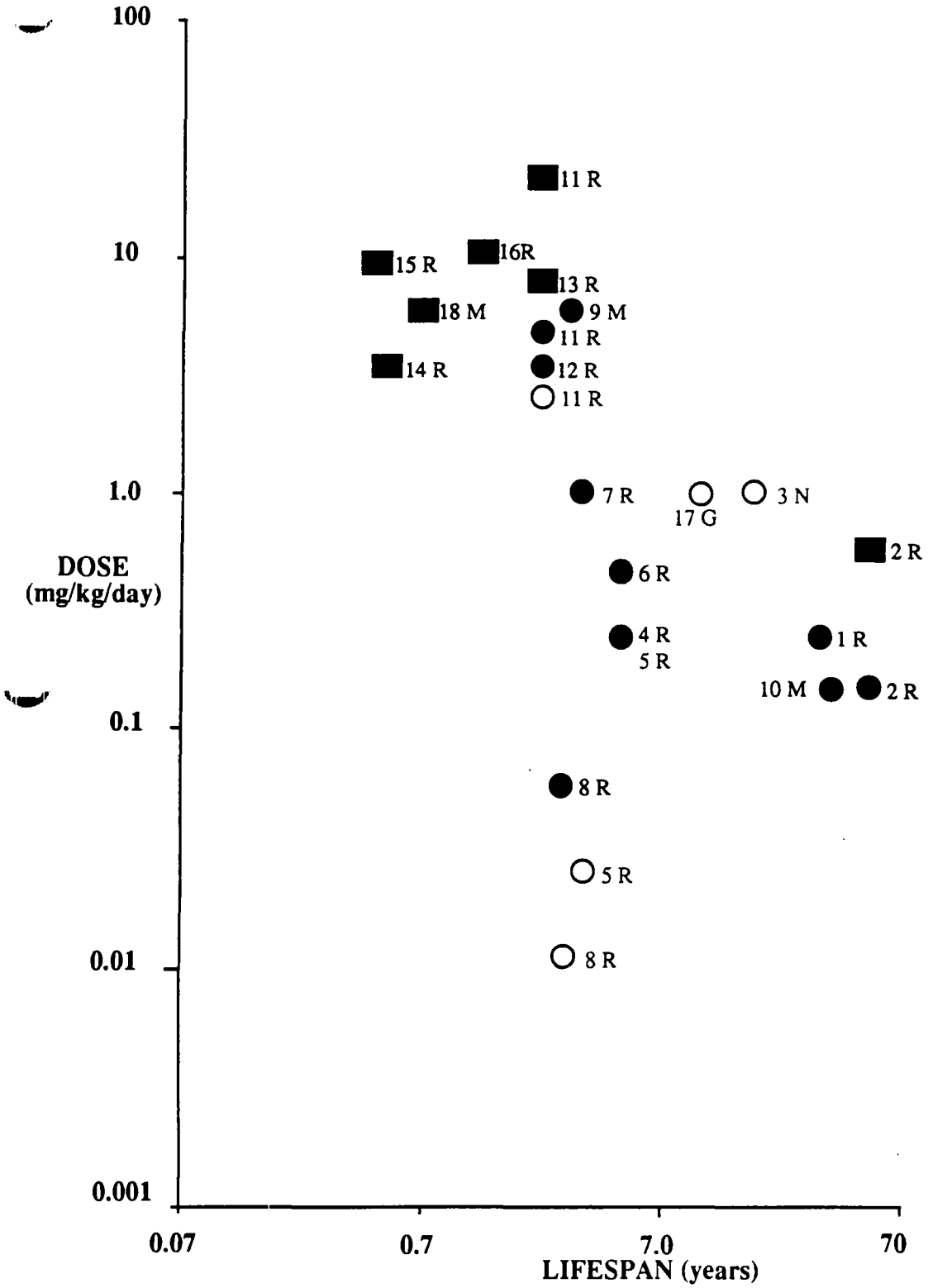
FIGURE KEY	SPECIES	ROUTE	EXPOSURE FREQUENCY/ DURATION	EFFECT	NOAEL (mg/kg/day)	LOAEL (Effect) LESS SERIOUS (mg/kg/day)	LOAEL (Effect) SERIOUS (mg/kg/day)	REFERENCE
1	Rat/SD	Diet	8 or 12 mo	Liver	—	0.25	—	Fulfs et al. 1977
2	Mouse/CD-1	Diet	≤ 18 mo	Liver	—	0.13	0.65	Fulfs et al. 1977
3	Monkey/Rhesus	Gavage	≤ 26 mo	Liver	1.0	—	—	Fulfs et al. 1977
4	Rat/SD	Diet	28 d	Liver, Thyroid	—	0.25	—	Singh et al. 1979, 1985
5	Rat/SD	Diet	28 d	Liver, Thyroid	0.025	0.25	—	Yarbrough et al. 1981
6	Rat/SD	Diet	28 d	Liver	—	0.5	—	Abston and Yarbrough 1976
7	Rat/SD	Diet	15 d	Liver	—	1.0	—	Curtis and Hoyt 1984
8	Rat/SD	Diet	14 d	Liver	0.0125	0.05	—	Iverson 1976
9	Mouse/CD-1	Diet	14 d	Liver	—	7.8	—	Byard and Pittman 1975
10	Mouse/CD-1	Diet	1-70 wk	Liver	—	0.13	—	Byard et al. 1974, 1975
11	Rat/CD	Gavage	Gestation 7-16 d	Reprod/ Develop	5	7	19	Chernoff et al. 1979a

TABLE V-1 (continued)
LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO MIREX

FIGURE KEY	SPECIES	EXPOSURE ROUTE	EXPOSURE FREQUENCY/ DURATION	EFFECT	NOAEL (mg/kg/day)	LOAEL (Effect) LESS SERIOUS (mg/kg/day)	LOAEL (Effect) SERIOUS (mg/kg/day)	REFERENCE
12	Rat/CD	Oral	Gestation 7-16 d	Reprod/ Develop	—	6	—	Kavlock et al. 1982
13	Rat/CD	NR	Gestation 5-14 d	Reprod/ Develop	—	—	10	Byrd et al. 1980
14	Rat/Sherman	Gavage	Postpartum 1-5 d	Reprod/ Develop	—	—	5	Scotti et al. 1981
15	Rat/Long-Evans	Gavage	Postpartum 1-4 d	Reprod/ Develop	—	—	10	Rogers and Grabowski 1984
16	Rat/CD	Oral	Gestation Various intervals	Reprod/ Develop	—	—	10	Buelke-Sam et al. 1983
17	Goat	Oral	1 mg/kg/2-61 wk 10 mg/kg/2-4 wk	Reprod/ Develop	1/10	—	—	Smrek et al. 1977
18	Mouse/CD-1	Oral	Gestation 8-14 d	Reprod/ Develop	—	—	7.5	Chernoff and Kavlock 1982

NR = Not Reported

FIGURE V-1
EFFECT-DOSE-DURATION FOR MIREX ANIMAL STUDIES



The data points plotted above correspond with the individual studies summarized in Table V-1. Numbers refer to figure key and R, M, N, and G refer to rat, mouse, monkey, and goat respectively. Effect levels are indicated by the following symbols: ■ = LOAEL (serious effects); ● = LOAEL (less serious effects); ○ = NOAEL. It should be noted that, in some cases, LOAELs for less serious effects may actually be more appropriately characterized as NOAELs and that some NOAELs may be more appropriately characterized as NOELs. Dose durations are divided by the appropriate species lifespan to yield a fraction which, when multipled by 70 years (the assumed average human lifespan), gives the corresponding position on the X-axis.

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Technical Support for Evaluating the Carcinogenic Potential of Mirex

ENVIRON

TECHNICAL SUPPORT FOR EVALUATING THE CARCINOGENIC POTENTIAL OF MIREX

INTRODUCTION

With minor exceptions, use of mirex-containing pesticides was effectively prohibited by the mid-1970s. Consequently, the Environmental Protection Agency (EPA) has not provided a verified cancer slope for mirex (CAS 2385-85-5) in its Integrated Risk Information System (IRIS). However, EPA did review the available carcinogenicity data on mirex in its 1987 Health Effects Assessment for Mirex, and classified it in EPA's "Weight-of-Evidence Group B2, probable human carcinogen." The EPA based its determination on what the Agency considered to be sufficient evidence from animal studies (multiple experiments in different species) and inadequate evidence from human studies. In the Health Effects Assessment, EPA recommended an oral cancer slope factor of 1.8 $(mg/kg/d)^{-1}$, based upon a draft National Toxicology Program (NTP) cancer bioassay for this compound (NTP 1987).

It is our concern that an estimate of the potential carcinogenicity of mirex based upon the published NTP bioassay results is not supportable because of changes in the diagnostic criteria and terminology for liver lesions that have taken place within the NTP since the mirex study was evaluated in 1983, and because information concerning cancer incidence rates in NTP historical control animals has been revised since preparation of the NTP bioassay report. Prior to 1975, there was no standardized nomenclature for hepatoproliferative lesions in the rat and evaluation of the potential carcinogenicity of a chemical could be highly dependent on an individual pathologist's terminology for these lesions. For example the terms "nodular hyperplasia, hepatoma, and carcinoma" could all potentially be used to characterize the same lesion. In 1975, Squire and Levitt proposed that the term "neoplastic nodule" be used to describe a variety of histological changes observed in rodent bioassays as a way of conservatively including any and all histological changes that may be associated with tumorigenesis in dosed animals. The National Academy of Sciences (NAS) essentially accepted the suggestions of Squire and Levitt (1975) and published a comprehensive monograph on this topic (ILAR 1980). Pathologists reading rodent slides from NTP studies conducted from the mid-1970s until the mid-1980s applied the criteria recommended in the NAS monograph.

After the NTP had finished a number of carcinogenicity studies of compounds in the 1980s, it became apparent that the terminology used to describe liver lesions was leading to regulatory difficulties. Maranpot et al. (1986) summarized the controversy as follows:

> Objection to the term neoplastic nodule generally focuses on three issues. The first relates to the connotation of neoplastic in the diagnosis. At issue here is the concept that an hepatic nodule, i.e., a focal proliferation of parenchymal cells, is not necessarily a neoplasm just because the accepted terminology contains the qualifier neoplastic. The second objection relates to the regulatory consequences of considering neoplastic nodules as true neoplasms.

> The third objection focuses on the obvious departure from conventional pathologic nomenclature. Use of the term neoplastic nodule for rat hepatoproliferative lesions, then, suggests that there is some compelling reason for departing from conventional pathologic nomenclature. Such a reason is not now apparent.

The Maranpot (1986) paper was published as representing NTP policy, and recommended the classification of focal areas of hyperplasia in rat liver as hyperplastic foci, or as benign adenomas, rather than as neoplastic nodules. The current understanding of the potential tumorigenicity of hepatocellular proliferative lesions as reflected in the terminology now in use could have an impact on interpretation of such lesions, e.g., in determination of the numbers of tumors found in a study, as compared to the terminology used in the past.

In its evaluation of some of the neoplasias observed in the mirex bioassay (e.g., pheochromocytomas and leukemias), the NTP (1990; the final version of the NTP 1987 report) considered the background incidences of these lesions in historical control animals. However, since preparation of the mirex report by NTP, the NTP historical control database has been revised to remove inappropriate data or data not meeting minimum quality control objectives. Accordingly, conclusions about the incidences of tumors over background levels, based on consideration of this information prior to its revision, may require reconsideration. The calculations of a revised oral cancer slope factor for mirex included in the subsequent portions of this document are based, in part, on consideration of these revised background incidence values.

This petition explains why EPA's weight-of-evidence classification and its oral cancer slope factor for mirex require reevaluation. It first describes the scientific data available for evaluating the potential carcinogenicity of mirex. It next describes the EPA's rationale for calculating a slope factor for mirex, and presents a weight-of-evidence evaluation of the potential carcinogenicity of mirex. Finally, this petition presents alternative slope factor calculations and recommendations for characterizing the potential carcinogenicity of mirex; it is our conclusion that these approaches provide superior and scientifically defensible bases for characterizing potential risks to the public health associated with environmental exposures to this compound.

REVIEW OF THE AVAILABLE CARCINOGENICITY DATA

This section summarizes the human and animal data available for evaluating the potential carcinogenicity of mirex. It includes discussions of one epidemiology study (Janerich et al. 1981), one study in mice (reported in Innes et al. 1969, and NCI 1968), and two studies in

rats (Ulland et al. 1977; NTP 1990, and a reevaluation of this study by PWG 1992). A weight-of-evidence evaluation of the data from these studies is presented in the next section of this petition.

Human Data

The only epidemiological study of potential cancer incidences related to mirex exposure appears to be an investigation of residents of the Love Canal area in New York (Janerich et al. 1981; EPA 1987). Janerich et al. (1981) found no evidence that elevated cancer rates were associated with residence in this area, and, consequently, found no evidence of human cancers associated with exposure to mirex.

Experimental Data

Innes et al. (1969). Innes et al. (1969, also reported in NCI 1968) tested commercial-grade mirex (98% pure) in two hybrid strains of mice, (C57BL/6 x C3H/Anf)F1 and (C57BL/6 x AKR)F1. The Innes et al. (1969) publication also contained evaluations of potential carcinogenicity of a number of other pesticides and industrial chemicals. In the mirex portion of the study, groups of 18 male and 18 female mice of each strain received 10 mg/kg body weight by gavage daily for 3 weeks, beginning at 7 days of age. The mice then received a diet containing 26 ppm (approximately 10 mg/kg body weight per day) mirex for the remaining portion of the study. This exposure regimen was intended to approximate the maximum tolerated dose. The scheduled study duration was 18 months; however, all the test mice died by 70 weeks.

As reported by Innes et al. (1969), mirex caused increased incidences of hepatomas (at either the $p < 0.05$ or $p < 0.01$ level) in both strains and sexes of the mice tested (Table 1). The hepatoma classification used in the Innes et al. (1969) report included both benign and malignant liver tumors. It should be noted that liver carcinomas reportedly were rarely observed in any of the individual experiments conducted as part of this study.

In a companion experiment (NCI 1968) with the same strains and numbers of mice as used in the Innes et al. (1969) study, 4-week-old mice received a single 1,000 mg mirex/kg body weight subcutaneous injection of mirex in 0.5% gelatin. Control groups of 18 mice each included untreated animals, and animals injected only with the gelatin vehicle, corn oil, or dimethylsulfoxide. More than 80% of the test and control mice survived to the end of the

study (i.e., 78 weeks). At necropsy, the combined incidences of sarcomas (reticulum cell carcinomas) in the two strains and sexes, as well as the combined incidences of hepatomas in the test animals, were higher than those of the controls (Table 2). However, these results were not statistically significant when only sex or vehicle matched controls were considered.

Ulland et al. (1977). Ulland et al. (1977) administered commercial-grade mirex (99% pure) in feed to Charles River CD rats for 18 months. Groups of 26 rats of each sex received mirex at 40 and 80 ppm in the feed for 9 weeks. In the tenth week, the dose levels were increased to 50 and 100 ppm, and were maintained at this level until the end of the 18-month study period. The high and low dose rates used in the study were intended to approximate the maximum tolerated dose and one-half the maximum tolerated dose, respectively. In addition, 20 untreated rats of each sex served as controls. All rats were necropsied and received thorough histopathological examinations. Surviving rats were sacrificed 24 months after the beginning of the dosing period. Survival rates in all but the low dose females were decreased, suggesting dose-related, and possibly sex-related, toxic effects.

A wide variety of liver changes was observed in the Ulland et al. (1977) study, including fatty metamorphosis and megalocytosis of hepatocytes, cystic degeneration and necrosis, biliary hyperplasia with periportal fibrosis, circumscribed areas of cellular alteration, neoplastic nodules, and carcinomas (Table 3). The incidence of neoplastic nodules was elevated only in the high-dose males ($p < 0.05$). In three of the four treatment groups, at least one of the animals with neoplastic nodules also had a hepatocellular carcinoma.

In their report, Ulland et al. (1977) noted that a preliminary evaluation of their data (Ulland et al. 1973) had indicated that mirex did not invoke a carcinogenic response. After reclassifying the observed liver lesions according to the then new guidelines established by a National Cancer Institute workshop on rat liver tumors (Squire and Levitt 1975), however, the authors (Ulland et al. 1977) concluded that the spectrum of observed liver lesions did suggest carcinogenic activity for this compound. It should be noted that the guidelines for classifying rat liver tumors have subsequently been revised again since the publication of the 1977 study. The new classification scheme appears to reflect more closely the scheme used by Ulland et al. in their 1973 preliminary evaluation of this bioassay.

National Toxicology Program (1990). The EPA, in its Health Effects Assessment for Mirex (EPA 1987), focused its quantitative evaluation of the potential carcinogenicity of mirex on data from a draft report of the NTP cancer bioassay (NTP 1987) for this compound. The final version of this study, which was published in 1990, does not appear to differ substantially from the 1987 draft, based on the data reported in the EPA Health Effects Assessment (EPA 1987). Accordingly, data from the 1990 publication of the NTP study are summarized in this petition. In the NTP bioassay (NTP 1990), groups of 52 F344/N rats of

each sex were fed diets containing 0, 0.1, 1.0, 10, 25, or 50 ppm of mirex for 104 weeks. Because of the high survival rates and the absence of any observable toxic effects in female rats during the first 6 months of this study, a second study was begun in which groups of 52 female F344/N rats were fed dietary concentrations of 0, 50, or 100 ppm mirex. Surviving animals from both studies were sacrificed 8 to 10 weeks after the last dosing week.

Mean body weights of male rats that received 25 or 50 ppm mirex were 5-18% lower than those of the controls throughout most of the study; mean body weights of female rats that received 50 or 100 ppm mirex were 4-18% lower than those of the controls after week 40; mean body weights of the other test groups were similar to those of controls. At the end of the study, overall survival of male rats that received 25 or 50 ppm mirex was lower than that of the controls ($p < 0.001$). Survival rates of the remaining male test groups and all the female test groups were similar to those of the controls.

The NTP report concluded that the most notable compound-related effects were observed in the livers of male and female rats. In the male rat study and in the first and second female rat studies, there were dose-related increases in the numbers of rats with non-neoplastic liver lesions. These lesions included fatty metamorphosis, cytomegaly, angiectasis (males only), and necrosis. The incidences of neoplastic nodules of the liver were dose-related and, in the 10-, 25-, and 50-ppm groups of males and in the 50- and 100-ppm groups of females (second study), were higher ($p < 0.01$) than in the controls. The incidences of hepatocellular carcinomas in the control and test groups were relatively low, and were not significantly different between groups (Tables 4-6).

Benign pheochromocytomas of the adrenal gland occurred with a positive trend and were higher than controls ($p < 0.05$) in the 25- and 50-ppm male rats (Table 4). Malignant pheochromocytomas were observed in two controls and in two mirex-exposed male rats. In females, the incidence of these combined lesions was marginally greater ($p > 0.05$) than that in the controls only in the 50-ppm female rats in the first study. These lesions in females were not considered to be due to the dietary administration of mirex.

Transitional cell papillomas of the kidneys occurred with a positive, dose-related trend in male rats. However, the incidences of these tumors were not significantly elevated over controls by the incidental tumor test. The authors of the NTP report expressed doubts concerning the biological significance of these findings and over the ability of this tumor type to progress to a malignant form.

In both the first and second studies in female rats, the incidences of mononuclear cell leukemia showed dose-related increases (Tables 5-6). The incidences of mononuclear cell leukemia were higher ($p < 0.05$) in the 25- and 50-ppm groups in the first study and in the 100-ppm group in the second study. Because the incidences in the control groups in both studies were similar, combined statistical analyses were done; the positive trends remained,

and the 10-, 25-, 50-, and 100-ppm groups were reported to exhibit increased incidences ($p < 0.05$) compared with the controls.

The authors of the NTP report concluded that there was clear evidence of carcinogenic activity of mirex in male F344/N rats, as indicated primarily by marked increased incidences of benign neoplastic nodules of the liver, as well as by increased incidences of pheochromocytomas of the adrenal gland and transitional cell papillomas of the kidney, and in female F344/N rats, as indicated by the increased incidence of mononuclear cell leukemia in females.

Pathology Working Group (1992). As a result of extensive carcinogenesis testing and research in laboratory rodents during the last several years, the nomenclature and criteria for proliferative hepatocellular lesions have evolved in order to better reflect the nature of these lesions and the mechanisms for their induction (Eustis et al. 1990; ILAR 1980; Maronpot et al. 1986; NTP (1989); Squire and Levitt 1975). The guidelines currently in use for evaluation of these types of data have changed significantly since the histological slides from the 1990 NTP bioassay for mirex were originally read. Because mirex may be a pollutant of concern at some hazardous waste sites currently being evaluated, the interpretation of the NTP bioassay and the use of the tumor data from this study in quantitative risk assessment could affect decisions made with regard to protection of human health by government regulators. Accordingly, PATHCO, Inc. was asked to conduct an independent panel review of the liver slides from the mirex study in order to evaluate the liver lesions using current diagnostic criteria and terminology.

Based on a review of the slides from the 1990 NTP bioassay, the PATHCO, Inc. Pathology Working Group (PWG 1992, Appendix A) concluded that administration of mirex was associated with increased incidences of benign hepatocellular tumors (adenomas) in both sexes (Tables 4-6). However, the incidences of adenomas were considerably lower than those of the neoplastic nodules diagnosed by the original PWG for the NTP (1990) bioassay. The 1992 PWG did conclude that the number of animals with hepatocellular adenomas was increased ($p < 0.05$) in males in the 25- and 50-ppm treatment groups, and in females in the second study in the 50- and 100-ppm treatment groups. The PWG found no increase in the incidences of hepatocellular carcinomas in either sex.

The increased incidences of hepatocellular adenomas in both sexes were limited to doses of mirex that also induced hepatotoxicity, which was also evaluated by the PWG. Hepatotoxicity was also observed at doses lower than those associated with increased incidences of tumors. Hepatocellular adenomas in females were significantly increased ($p < 0.05$) in the second study, but not in the first study. The incidences of adenomas in the second study 50- and 100-ppm treatment groups were comparable. In addition, the actual numbers of adenomas in the 50-ppm treatment groups were not greatly different between the

two studies; however, the lack of adenomas in the second control group contributed to the observation of a significant elevation in the second study.

WEIGHT OF EVIDENCE FOR CARCINOGENICITY

This section provides a weight-of-evidence evaluation of the possible carcinogenicity of mirex in humans. The discussions presented in this section rely primarily on the studies summarized in the preceding section of this report. This section first outlines the conclusions regarding the potential carcinogenicity of mirex reached by the EPA in its 1987 Health Effects Assessment, and then provides an alternative analysis that takes into account information that has become available since the preparation of EPA's 1987 report. The analyses presented in this section were carried out within the framework of weight-of-evidence guidelines currently used by government regulatory agencies (e.g., EPA 1986; NTP 1990, p. 6; OSTP 1984).

Environmental Protection Agency (1987) Evaluation

In a discussion of the weight of evidence for carcinogenicity of mirex, the EPA Health Effects Assessment (EPA 1987) concluded that mirex should be classified in EPA Group B2, probable human carcinogen, based on sufficient evidence from animal studies (multiple experiments in different species) and inadequate evidence from human studies.

The studies considered by the EPA in its evaluation included the Innes et al. (1969) study in mice, the Ulland et al. (1977) study in rats, and the draft NTP (1987) bioassay in rats. In selecting data sets suitable for quantitative risk assessment, EPA (1987) concluded that the single dose level and high mortality in the Innes et al. (1969) study, the controversy concerning tumor classification in the Ulland et al. (1977) study, and the reporting inadequacies in both studies made these reports "less than optimum for quantitative risk assessment." EPA (1987) concluded that the draft NTP (1987) bioassay was not subject to these same deficiencies, and based its derivation of a mirex cancer slope factor on data from the study.

EPA (1987) estimated mirex cancer slope factors based on three different data sets derived from the draft NTP (1987) bioassay: (1) pooled counts for male rats with liver neoplastic nodules, hepatocellular carcinomas, adrenal pheochromocytomas, or malignant pheochromocytomas; (2) pooled counts for first study female rats with liver neoplastic

nodules, hepatocellular carcinomas, or leukemias; and (3) pooled counts for second study female rats with liver neoplastic nodules, hepatocellular carcinomas, or leukemias. These data purposefully included all benign and malignant tumor types for sites showing significantly elevated tumor incidences. EPA (1987) used the linearized multistage model, Global 82 (Howe and Crump 1982), to extrapolate the tumor data to low doses and projected 95% upper confidence limit human cancer slopes factors (q_1^*) of 1.8, 0.8, and 1.2 $(mg/kg/day)^{-1}$, respectively, for the three data sets. EPA (1987) selected the value of 1.8 $(mg/kg/day)^{-1}$ as a conservative estimate of the human cancer slope factor for mirex.

Alternative Evaluation

The weight-of-evidence evaluation presented in this section takes into consideration information that was not available to the EPA during the preparation of its 1987 Health Effects Assessment. The information available for evaluation, in addition to that considered by the EPA, primarily includes the recent PWG (1992) reevaluation of liver lesions in the NTP (1990) bioassay for mirex and more appropriate NTP historical control data for other types of lesions.

Janerich et al. (1981). Janerich et al. (1981) provided the only epidemiological study on cancer incidence related to mirex exposures. This study involved an investigation of the Love Canal area of New York and found no evidence of elevated cancer rates in residents of the area. However, the study population was small and there was an uncertain latency between first exposure to mirex and expected appearance of any disease.

Innes et al. (1969). The scientific value of the Innes et al. (1969) mirex feeding study in two strains of mice is limited due to the small sizes of the test groups (18 animals per group), the use of only one test dose (a dose which appeared to have exceeded the maximum tolerated dose because of high mortalities in both strains of mice), and general reporting inadequacies. In addition, it appears that the liver tumors identified in the study were primarily, or possibly exclusively, benign adenomas. Finally, it appears that the diagnostic criteria and terminology used in performing the pathological evaluations for this study probably resulted in the classification of some lesions as being neoplastic, but which would be considered to be hyperplastic by NTP's current guidelines. It is very likely that the use of current diagnostic criteria and terminology, as defined by NTP, would result in appreciably fewer total tumors. Given these shortcomings and uncertainties, this study provides only equivocal evidence of carcinogenic activity, as defined by the NTP (1990, p. 6). It is not suitable for quantitative risk assessment.

National Cancer Institute (1968). The mirex subcutaneous injection study in two strains of mice (NCI 1968) suffers from many of the same deficiencies as its companion feeding study (Innes et al. 1969), cited above. These include the use of small test groups (18 animals per group), the use of diagnostic criteria and terminology for liver tumors that have been revised since publication of the study, and general reporting inadequacies. In addition, the lack of statistically significant elevations of liver tumors and reticulum cell carcinomas in test groups, when compared to matched controls, limits the ability of this study to support a positive finding. Finally, the relevance of the subcutaneous route of exposure to expected human exposure routes is questionable. Given these shortcomings and uncertainties, this study provides only equivocal evidence of carcinogenic activity, as defined by the NTP (1990, p. 6). It is not suitable for quantitative risk assessment.

Ulland et al. (1977). The scientific value of the Ulland et al. (1977) mirex feeding study in Charles River CD rats is limited due to the small sizes of the test groups (26 animals per test group, 20 per control group), the high mortalities of the test groups, the apparent high toxicity of the administered doses, and general reporting inadequacies. In addition, it is likely that a reevaluation of the liver tumors identified in these animals, using current diagnostic criteria and terminology, would result in appreciably fewer total tumors. A preliminary report of this study (Ulland et al. 1973) did not identify significantly increased incidences of liver tumors using different diagnostic criteria than those ultimately used in the 1977 study. NTP's current classification scheme appears to reflect more closely the scheme used by Ulland et al. in their 1973 preliminary evaluation of this bioassay. Nevertheless, it should be noted that a few hepatocellular carcinomas were identified in the test animals, but not the controls. However, the incidence of this tumor was not significantly elevated. Given these shortcomings and uncertainties, this study provides only equivocal evidence of carcinogenic activity, as defined by the NTP (1990, p. 6). It is not suitable for quantitative risk assessment.

National Toxicology Program (1990). Based on the PWG (1992) review of slides with hepatocellular proliferative lesions, data from the NTP (1990) study indicated that administration of mirex was associated with increased incidences of benign hepatocellular tumors (adenomas) in male and female F344/N rats. There was no increase in the incidences of hepatocellular carcinomas in either sex. The number of treated male rats with hepatocellular adenomas was statistically elevated in the 25- and 50-ppm dose groups. The number of treated female rats with hepatocellular adenomas was statistically significant only in the 50- and 100-ppm dose groups of the second study. The incidences of adenomas in these two groups were comparable, indicating the lack of a positive dose response relationship. These incidences also were comparable with the incidence of hepatocellular adenomas in the first study 50-ppm group. The lack of adenomas in the controls of the

second study contributed to the significance observed for hepatocellular adenomas in females. Conversely, the incidence of hepatocellular tumors in the control animals in the first study was approximately two times higher (6% versus 2.9%) than the mean historical incidence reported by NTP (1990).

The increased incidences of hepatocellular adenomas in both sexes were limited to animals receiving doses of mirex that also induced hepatotoxicity and eosinophilic foci. Hepatotoxicity was also observed at doses lower than those with increased incidences of tumors. Whereas mirex-induced toxicity exhibited strong dose response relationships at doses up to 25 ppm, no further increases in toxicity occurred at the higher doses in males or females. This information suggests that extensive mirex-induced cell damage accompanied by increased cell turnover (replication, proliferation, mitogenesis) may be necessary for this compound to be associated with any type of tumorigenic (e.g., promotional) activity. Levels not producing extensive toxicity would not be expected to affect tumor incidences.

The incidences of benign pheochromocytomas of the adrenal gland showed a positive dose trend in male rats, and the incidences in the 25- and 50-ppm groups were significantly higher than those in controls (NTP 1990). Malignant pheochromocytomas were observed in two control rats and in two mirex-exposed rats. NTP (1990) considered the magnitude and dose response of these lesions in males sufficient to make an association with mirex administration. It was noted by the NTP that the control incidence of pheochromocytomas (16%) agreed well with the mean historical incidence for untreated male rats (22%). However, the control incidence in the mirex study (16%) is somewhat lower than the historical incidence (22%) reported by NTP (1990), and appreciably lower than contemporaneous historical incidences of 30-34% to >40% reported for F344/N rats in other sources (NTP 1992, IARC 1976, Solleveld 1984). These historical rates are comparable to the pheochromocytoma incidence rates observed in the 25- and 50-ppm male rat groups. The historical control data reported in the 1990 NTP bioassay report have been subsequently revised by NTP to include only those data that have undergone appropriate quality control review. These more recent data (NTP 1992) should supersede the data presented in the 1990 NTP bioassay report. Consideration of the historical control rates for this common tumor in male F344/N rats places uncertainty on the sufficiency of these results for demonstrating an association between mirex administration and increased incidences of pheochromocytomas. In addition, it should be noted that elevated incidences of pheochromocytomas have not been reported in any other experimental studies of mirex. Taken together, the findings in the NTP (1990) report appear to be insufficient to relate the incidences of pheochromocytoma, a common lesion in male F344/N rats, with mirex administration.

Mononuclear cell leukemia showed positive dose-related trends in the first and second studies in female rats. Increased incidences of leukemia were observed in the 25- and 50-ppm groups in the first study, and in the 100-ppm group in the second study. NTP (1990) concluded that the association of mononuclear cell leukemia with mirex administration was

indicated "primarily because the rather marginal increases occurred in both studies." NTP (1990) combined the two studies in females, because the incidences of leukemia did not differ statistically between the two control groups (15% and 12%), and found that the incidences in the 10-, 25-, 50-, and 100-ppm groups were greater than the combined control incidence. It should be noted, however, that the percentage of female rats with leukemia in the second study 50-ppm group (17%) was comparable to the percentages in the first study control, 0.1-ppm, and 1-ppm groups (15%, 15%, 21%), and that the percentage in the second study 100-ppm group (27%) was comparable to the percentage in the first study 10-ppm group (27%). Thus, the incidences reported in the first-study lower dose groups were not consistent with the incidences in the second study test groups. Furthermore, the control incidences in these experiments (15 and 12%) are somewhat lower than the historical incidence (19%) reported by NTP (1990), and appreciably lower than contemporaneous historical incidences of 24-26% or higher, as reported for F344/N rats in other sources (NTP 1992, IARC 1973, Solleveld 1984). As stated earlier, the historical control data reported in the 1990 NTP bioassay report have been revised to include only those data that have undergone appropriate quality control review. These more recent data (NTP 1992) should supersede the data presented in the 1990 NTP bioassay report. Finally, excess incidences of leukemias have not been reported in any other experimental studies of mirex. Taken together, the results of the NTP (1990) study are insufficient to relate the incidences of mononuclear cell leukemia, a common lesion in female F344/N rats, with mirex administration.

Overall, the results of the NTP (1990) study provide some evidence of carcinogenic activity as defined by NTP (1990, p. 6). There was a mirex-related increase of benign hepatocellular adenomas, as characterized by the 1992 PWG, in test animals that exhibited extensive and severe mirex-related liver damage. However, the incidences of these tumors were not markedly increased. Additionally, as discussed above, the incidences of pheochromocytomas in males and leukemia in females were reported to be increased over those of controls in the NTP (1990) study. However, these results do not appear to provide sufficient evidence that the observed neoplasias were related to mirex administration. These neoplasias occur commonly and at variable rates in F344/N rats, and these findings have not been replicated in other studies of mirex. Although these tumors occurred more frequently in some dose groups than in controls in this study, the reported incidences in the mirex-exposed groups were comparable to historical control incidences (NTP 1992). Furthermore, high variability in tumor incidences was observed for the leukemias within the NTP (1990) study; the incidences reported for the first female rat study were not consistent with the incidences reported for the second study.

Recommended Weight of Evidence Classification

Consideration of the available data on the potential carcinogenicity of mirex suggests that this compound could potentially be classified in EPA weight-of-evidence Group B2, probable human carcinogen, or in Group C, possible human carcinogen. The Group B2 and Group C classifications are based on findings of inadequate evidence for carcinogenicity in human studies, and either sufficient or limited evidence, respectively, for carcinogenicity in animal studies.

Information that is normally required to provide sufficient evidence for carcinogenicity, and a Group B2 classification, includes the finding of malignant, or combined malignant and benign tumors in multiple species, strains, or experiments. However, except for the leukemias reported in female rats in the 1990 NTP study, tumors potentially associated with exposures to mirex have been predominantly benign. Because leukemia is a commonly occurring cancer in F344/N rats, can occur at widely variable rates, occurred at rates comparable to historical controls, and did not occur with consistent elevations in the first and second NTP female rat studies, the incidences reported in the NTP bioassay should not be considered significantly elevated. Furthermore, the mouse and rat bioassays reported by Innes et al. (1969) and Ulland et al. (1977) have experimental design and reporting deficiencies that severely limit their usefulness.

Information that is normally required to provide limited evidence for the carcinogenicity of mirex, and a Group C classification, includes a malignant tumor response (i.e., leukemia) in a single well-conducted experiment that does not meet conditions for sufficient evidence; tumor responses of marginal statistical significance in studies having inadequate design or reporting (i.e., Innes et al 1969, Ulland et al. 1977); benign but not malignant tumors (hepatocellular adenomas, pheochromocytomas) with an agent showing no response in a variety of short-term tests for mutagenicity (i.e., mirex, as reported in NTP 1990); and responses of marginal statistical significance in a tissue known to have a high or variable background rate (i.e., leukemias, pheochromocytomas).

Based upon consideration of the issues detailed above, mirex is most appropriately classified in EPA's "Weight-of-Evidence Group C," as a possible human carcinogen.

QUANTITATIVE DOSE RESPONSE EVALUATION

Cancer slope factor estimates (95% upper confidence limit values) were developed using the linearized multistage model recommended for use in dose response modeling by the EPA

(Crump et al. 1991). All assumptions used in carrying out the dose response modeling were based on guidelines and procedures typically used by the EPA, with the exception of the interspecies scaling factor. In this dose-response modeling effort, actual doses in the test species were converted into estimated doses in the target species by assuming that doses were proportional to body weight raised to the three-fourths power. This assumption corresponds with current EPA proposed recommendations in this regard. Seven different data sets from the combined NTP (1990) and PWG (1992) reports were evaluated (Tables 7a-7g).

For male rats, three data sets were considered (derived from NTP 1990, Appendix A and PWG 1992, Appendix A): (1) animals having a hepatocellular adenoma, hepatocellular carcinoma, adrenal pheochromocytoma, or malignant pheochromocytoma (Table 7a); (2) animals having a hepatocellular adenoma or hepatocellular carcinoma (Table 7b); and (3) animals having a hepatocellular carcinoma (Table 7c). The cancer slopes calculated from these three data sets were 0.72, 0.34, and 0.085 $(mg/kg/d)^{-1}$, respectively. It should be noted that the excess cancer risks characterized by these values are due primarily to benign tumors found in the liver or adrenals. Only the cancer slope factor of 0.34 $(mg/kg/d)^{-1}$, based on data set 2, is considered to be appropriate for quantitative risk assessment under current EPA guidelines. As noted in the alternative weight-of-evidence analysis presented in the preceding section, a reevaluation of the available historical control data for pheochromocytoma incidences in male rats and the lack of reports of elevated incidences of these tumors in other mirex bioassays, suggests that these tumors are not associated with exposures to mirex. The cancer slope factor based on data set 1, which includes these tumors, is provided only for comparison. With regard to data set 3, there currently is scientific evidence to suggest that rodent benign hepatocellular adenomas typically would not be expected to progress to malignant hepatocellular carcinomas. Accordingly, data set 3 was used to calculate a cancer slope factor based only on the incidence of hepatocellular carcinomas. Nevertheless, because this scientific issue has not yet been completely resolved in a regulatory sense and because the EPA (1986) has recommended that benign tumors should generally be combined with malignant tumors for risk estimates, the cancer slope factor based on data set 3 also is provided only for comparison.

For female rats, four data sets were considered (derived from NTP 1990, Appendix B and PWG 1992, Appendix A): (1) animals in the first study having leukemia (Table 7d); (2) animals in the second study having a hepatocellular adenoma, hepatocellular carcinoma, or leukemia (Table 7e); (3) animals from the second study having a hepatocellular adenoma or hepatocellular carcinoma (Table 7f); and (4) animals having a hepatocellular carcinoma (Table 7g). The cancer slopes calculated from these four data sets were 0.54, 0.28, 0.13, and 0.024 $(mg/kg/d)^{-1}$, respectively. It should be noted that any portions of these excess cancer risks associated with liver lesions were due primarily to benign tumors. Only the cancer slope factor of 0.13 $(mg/kg/d)^{-1}$, based on data set 3, is considered to be appropriate for quantitative risk assessment. As noted in the alternative weight-of-evidence analysis presented in the preceding section, a reevaluation of the available historical control data for

leukemia incidences in female rats, the lack of reports of elevated incidences of these tumors in other mirex bioassays, and the inconsistent results for these tumors in the first and second NTP studies for female rats suggests that these tumors are not associated with exposures to mirex. The cancer slope factors based on data sets 1 and 2, which include these tumors, are provided only for comparison. With regard to data set 4, there currently is scientific evidence to suggest that rodent benign hepatocellular adenomas typically would not be expected to progress to malignant hepatocellular carcinomas. Accordingly, data set 4 was used to calculate a cancer slope factor based only on the incidence of hepatocellular carcinomas. Nevertheless, because this issue has not been completely resolved and because the EPA (1986) has recommended that benign tumors should generally be combined with malignant tumors for risk estimates, the cancer slope factor based on data set 4 also is provided only for comparison.

The estimate of 0.34 $(mg/kg/d)^{-1}$ is recommended to represent the best estimate of carcinogenic potential of mirex that can be made at this time. As discussed above, the available cancer bioassay data do not provide sufficient evidence that the pheochromocytomas and leukemias (tumors commonly occurring in unexposed controls) observed in the NTP (1990) study were related to mirex exposures. Conversely, a number of experimental studies have indicated that the liver is the primary target organ for high-dose mirex toxicity. In addition, liver tumors have been observed in more than one cancer bioassay. Thus, the highest cancer slope based on liver neoplasia in the NTP (1990) bioassay, as characterized by PWG (1992), was selected. Although this value is based primarily on benign tumors, it has been suggested by some scientists that these lesions may progress to carcinomas, and there is insufficient scientific knowledge at this time to definitively conclude otherwise. Accordingly, as recommended by EPA, pooled counts for benign and malignant tumors were used in this evaluation, leading to the recommended cancer slope factor of 0.34 $(mg/kg/d)^{-1}$.

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TABLE 1

INCIDENCE OF HEPATOMAS IN TWO STRAINS OF MICE CHRONICALLY EXPOSED TO MIREX IN THE DIET (10 mg/kg)

	CONTROLS		TREATED	
HYBRID	MALE	FEMALE	MALE	FEMALE
Strain X[a]	8/79	0/87	6/18*	8/16**
Strain Y[b]	5/90	1/82	5/15**	10/16**

[a] Strain X = (C57BL/6 x C3H/Anf)
[b] Strain Y = (C57BL/6 x AKR)
* $p < 0.05$
** $p < 0.01$

Source: Innes et al. 1969

TABLE 2

INCIDENCE OF NEOPLASTIC LESIONS AFTER 18 MONTHS IN TWO STRAINS OF MICE ADMINISTERED SINGLE SUBCUTANEOUS DOSES OF MIREX (1000 mg/kg)

	STRAIN X[a]		STRAIN Y[b]		
	MALE	FEMALE	MALE	FEMALE	TOTAL[c]
Reticulum Cell Carcinoma					
Control					
Combined	8/141	1/154	0/161	5/157	14/613
Gelatin	2/16	0/18	0/18	1/18	3/70
Test	6/18	0/17	1/17	3/18	10/70*
Hepatoma					
Control					
Combined	9/141	0/154	1/161	0/157	10/613
Gelatin	1/16	0/18	1/18	0/18	2/70
Test	2/18	0/17	4/17	1/18	7/70*

[a] Strain X = (C57BL/6 x C3H/Anf).
[b] Strain Y = (C57BL/6 x AKR).
[c] Combined strains and sexes.
* $p < 0.05$, combined strains and sexes versus combined controls.

Source: NCI 1968

TABLE 3
INCIDENCE OF HEPATIC LESIONS IN CD RATS
CHRONICALLY EXPOSED TO MIREX IN DIET

	FOCI/AREAS OF CELLULAR ALTERATION			NEOPLASTIC NODULES			HEPATOCELLULAR CARCINOMA[a]		
	DOSE[b]								
SEX	0[c]	LOW	HIGH	0[c]	LOW	HIGH	0[c]	LOW	HIGH
MALE	3/20	6/26	10/26	0/20	2/26	7/26*	0/20	1/26	4/26
FEMALE	10/20	11/26	7/26	0/20	4/26	4/26	0/20	0/26	1/26

[a] All animals at risk that developed hepatocellular carcinomas also had neoplastic nodules.
[b] Dose levels were set at 40 and 80 ppm for the first 10 weeks of the study, after which they were increased to 50 and 100 ppm, respectively.
[c] Untreated controls.
* $p < 0.05$

Source: Ulland et al. 1977

TABLE 4

Tumor Incidence in Male F344/N Rats Reported By The National Toxicology Program, 1990[a]

Dose (ppm)	0	0.1	1.0	10	25	50
Liver Neoplastic Nodule[b]	3 (1)	5 (1)	5 (2)	14** (4)	15** (6*)	26** (10*)
Hepatocellular Carcinoma[c]	3 (3)	0 (0)	2 (2)	2 (1)	3 (2)	4 (3)
Adrenal Pheochromocytoma	8/51	7	13	11	18*/51	19*/51
Malignant Pheochromocytoma	2/51	0	0	1	0	1/51

[a] All values reported are based on 52 tissue samples unless otherwise noted (e.g., 8/51).
[b] Values in parentheses are hepatocellular adenomas diagnosed by the Pathology Working Group, 1992.
[c] Values in parentheses are hepatocellular carcinomas diagnosed by the Pathology Working Group, 1992.
* $p < 0.05$
** $p < 0.01$

TABLE 5

Tumor Incidence in Female F344/N Rats Reported By The National Toxicology Program, 1990 First Study[a]

Dose (ppm)	0	0.1	1.0	10	25	50
Liver Neoplastic Nodule[b]	10 (3)	5 (1)	4 (1)	5 (0)	9 (2)	7 (3)
Hepatocellular Carcinoma[c]	0 (0)	0 (0)	0 (0)	0 (0)	1 (1)	2 (1)
Leukemia	8	8	10	13	17*	14*

[a] All values reported are based on 52 tissue samples.
[b] Values in parentheses are hepatocellular adenomas diagnosed by the Pathology Working Group, 1992.
[c] Values in parentheses are hepatocellular carcinomas diagnosed by the Pathology Working Group, 1992.
* $p < 0.05$

TABLE 6

Tumor Incidence in Female F344/N Rats Reported By The National Toxicology Program, 1990 Second Study[a]

Dose (ppm)	0	50	100
Liver Neoplastic Nodule[b]	2 (0)	23** (5*)	30** (5*)
Hepatocellular Carcinoma[c]	0 (0)	0 (0)	1 (1)
Leukemia	5	6	12*

[a] All values reported are based on 52 tissue samples.
[b] Values in parentheses are hepatocellular adenomas diagnosed by the Pathology Working Group, 1992.
[c] Values in parentheses are hepatocellular carcinomas diagnosed by the Pathology Working Group, 1992.
* $p < 0.05$
** $p < 0.01$

TABLE 7a

Cancer Data Sheet for Derivation of q_1*

Reference: NTP 1990, PWG 1992

Compound: mirex

Species, strain, sex: rat, F344/N, male

Route, vehicle: oral, diet

Length of exposure (le): 104 weeks

Length of experiment (Le): 104 weeks

Lifespan of animal (L): 104 weeks

Body weight: 0.35 kg

Tumor site and type: hepatocellular adenoma, hepatocellular carcinoma, adrenal pheochromocytoma, and malignant pheochromocytoma

Experimental Exposure (ppm)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested
0	0	14/51[a]
0.1	0.007	7/52
1	0.07	15/52
10	0.7	16/52
25	1.8	20/51[a]
50	3.8	27/52

[a] Adrenal not examined in one rat that did not have hepatocellular adenoma or hepatocellular carcinoma.

Human cancer slope = 0.72 $(mg/kg/day)^{-1}$

TABLE 7b

Cancer Data Sheet for Derivation of q_1*

Reference: NTP 1990, PWG 1992

Compound: mirex

Species, strain, sex: rat, F344/N, male

Route, vehicle: oral, diet

Length of exposure (le): 104 weeks

Length of experiment (Le): 104 weeks

Lifespan of animal (L): 104 weeks

Body weight: 0.35 kg

Tumor site and type: hepatocellular adenoma, hepatocellular carcinoma

Experimental Exposure (ppm)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested
0	0	4/52
0.1	0.007	1/52
1	0.07	4/52
10	0.7	5/52
25	1.8	7/52
50	3.8	13/52

Human cancer slope = 0.34 $(mg/kg/day)^{-1}$

TABLE 7c

Cancer Data Sheet for Derivation of q1*

Reference: NTP 1990, PWG 1992

Compound: mirex

Species, strain, sex: rat, F344/N, male

Route, vehicle: oral, diet

Length of exposure (le): 104 weeks

Length of experiment (Le): 104 weeks

Lifespan of animal (L): 104 weeks

Body weight: 0.35 kg

Tumor site and type: hepatocellular carcinoma

Experimental Exposure (ppm)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested
0	0	3/52
0.1	0.007	0/52
1	0.07	2/52
10	0.7	1/52
25	1.8	2/52
50	3.8	3/52

Human cancer slope = 0.085 $(mg/kg/day)^{-1}$

TABLE 7d

Cancer Data Sheet for Derivation of q_1*

Reference: NTP 1990, PWG 1992

Compound: mirex

Species, strain, sex: rat, F344/N, female (first study)

Route, vehicle: oral, diet

Length of exposure (le): 104 weeks

Length of experiment (Le): 104 weeks

Lifespan of animal (L): 104 weeks

Body weight: 0.23 kg

Tumor site and type: leukemia

Experimental Exposure (ppm)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested
0	0	8/50[a]
0.1	0.007	8/52
1	0.08	11/52
10	0.7	14/51[a]
25	2.0	18/51[a]
50	3.9	18/51[a]

[a] Spleen not examined in one or two rats that did not have leukemia in other unspecified organs.

Human cancer slope = 0.54 $(mg/kg/day)^{-1}$

TABLE 7e

Cancer Data Sheet for Derivation of q_1*.

Reference: NTP 1990, PWG 1992

Compound: mirex

Species, strain, sex: rat, F344/N, female (second study)

Route, vehicle: oral, diet

Length of exposure (le): 104 weeks

Length of experiment (Le): 104 weeks

Lifespan of animal (L): 104 weeks

Body weight: 0.23 kg

Tumor site and type: hepatocellular adenoma, hepatocellular carcinoma, leukemia

Experimental Exposure (ppm)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested
0	0	6/52
50	3.9	12/52
100	7.7	19/50[a]

[a] Spleen not examined in two rats that did not have hepatocellular adenoma, hepatocellular carcinoma, or leukemia in other unspecified organs.

Human cancer slope = 0.28 $(mg/kg/day)^{-1}$

TABLE 7f

Cancer Data Sheet for Derivation of q_1^*

Reference: NTP 1990, PWG 1992

Compound: mirex

Species, strain, sex: rat, F344/N, female (second study)

Route, vehicle: oral, diet

Length of exposure (le): 104 weeks

Length of experiment (Le): 104 weeks

Lifespan of animal (L): 104 weeks

Body weight: 0.23 kg

Tumor site and type: hepatocellular adenoma, hepatocellular carcinoma

Experimental Exposure (ppm)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested
0	0	0/52
50	3.9	5/52
100	7.7	6/52

Human cancer slope = 0.13 $(mg/kg/day)^{-1}$

TABLE 7g

Cancer Data Sheet for Derivation of q1*

Reference: NTP 1990, PWG 1992

Compound: mirex

Species, strain, sex: rat, F344/N, female (second study)

Route, vehicle: oral, diet

Length of exposure (le): 104 weeks

Length of experiment (Le): 104 weeks

Lifespan of animal (L): 104 weeks

Body weight: 0.23 kg

Tumor site and type: hepatocellular carcinoma

Experimental Exposure (ppm)	Transformed Dose (mg/kg/day)	Incidence No. Responding/No. Tested
0	0	0/52
50	3.9	0/52
100	7.7	1/52

Human cancer slope = 0.024 $(mg/kg/day)^{-1}$

APPENDIX E

A Review of Photomirex

A REVIEW OF PHOTOMIREX

February 23, 1993

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I. INTRODUCTION

Photomirex is not a commercial chemical but is the major photodegradation product of the insecticide mirex. Mirex is a fully chlorinated organic compound with the chemical formula $C_{10}Cl_{12}$, and was marketed commercially in the United States as an insecticide and fire-retardant additive from the late 1950s until the mid 1970s (Kaiser 1978). During the 1960s, mirex-containing baits were used extensively as insecticides to control fire ants in the southeastern United States. The bait were mixtures of mirex, soybean oil, and sometimes corn cob grit, which were delivered by aircraft, helicopter, or tractor (WHO 1984). From 1962 to 1976 about 226,000 kg of mirex in the form a fire ant bait was applied to 132,000,000 acres in 10 states (IARC 1979).

In 1971, based upon concerns about the potential environmental and toxicological properties of mirex, the United States Environmental Protection Agency (EPA) issued a pesticide cancellation order for mirex, pending release of an environmental impact study. The order was appealed, and in 1972 EPA reinstated all mirex registrations and issued new guidelines which placed some restrictions on the use of mirex in the control of fire ant populations (IARC 1979). Following renewed controversy over its potential carcinogenicity in 1976, EPA announced plans to phase out the use of mirex, and to cancel all registrations for products containing mirex as an active ingredient. With minor exceptions, the use of mirex-containing insecticides effectively ceased by mid-1978 (WHO 1984).

In the presence of ultraviolet radiation, some mirex will be reduced to photomirex. Because photomirex has been claimed by various authors to have similar chemical and physical properties to the parent chemical, concern has risen over levels of photomirex in the environment and its potential toxicological properties.

This review summarizes the identified scientific literature on photomirex, and is intended to provide a basis for evaluation of its potential human health effects at low levels of environmental exposures.

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II. PHYSICAL/CHEMICAL PROPERTIES

Photomirex (CAS No. 39801-14-4) is a chlorinated hydrocarbon breakdown product of mirex. It is known by a variety of synonyms, including hydromirex, 8-monohydromirex, and 1,2,3,4,5,5,6,7,9,10,10-undecachloropentacyclo[5.3.0.$0^{2,6}$.$0^{3,9}$.$0^{4,8}$]decane (Hallett et al. 1978, RTECS 1992). It has the chemical formula $C_{10}HCl_{11}$ (Figure II-1).

GENERAL PHYSICAL AND CHEMICAL PROPERTIES

Photomirex is produced upon the chemical reduction of mirex in the presence of ultaviolet radiation. Photomirex has similar chemical and physical properties to mirex (Kaiser 1978), which is extremely stable, practically insoluble in water (< 1 ppb), and has a very low vapor pressue (WHO 1984). Environmentally, mirex is tightly bound to organic matter in sediments and soils, and may persist in this state for prolonged periods (Kaiser 1978).

There are three possible monohydro derivatives of mirex, referred to as 8-, 9-, or 10-monohydromirex. Initially, Alley et al. (1973) were able to determine that 10-monohydromirex was not the photoproduct, but they were unable to distinguish if the product was 8-monohydromirex (photomirex) or 9-monohydromirex. However, by undertaking an investigation of the structures of photoproducts of the structurally related compound kepone, Alley et al. (1974) were able to unequivocally establish the photoproduct of mirex as being 8-monohydromirex (photomirex).

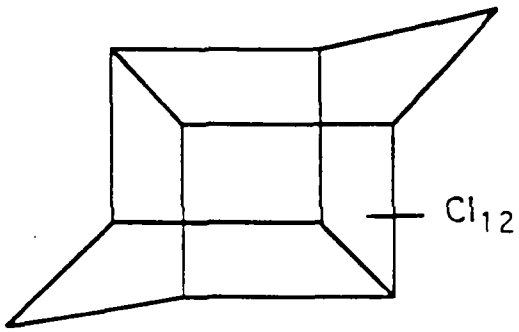
Mudambi and Hassett (1988) found that the photolysis of mirex in water is given by the following equation:

$$\text{MIREX} \Rightarrow \text{PHOTOMIREX} \Rightarrow \text{2,8-DIHYDROMIREX}$$

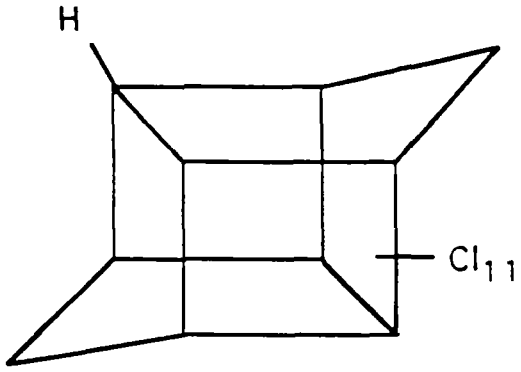
Mirex is reduced through the loss of one chlorine atom in exchange for a hydrogen atom; the resulting compound is photomirex. As shown in the above equation, photomirex can be further dichlorinated to form 2,8-dihydromirex. Environmental conditions determine the relative rates of degradation in each instance.

FIGURE II-1

CHEMICAL STRUCTURE OF MIREX AND PHOTOMIREX



Mirex



Photomirex

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III. ENVIRONMENTAL FATE AND BEHAVIOR

SOURCES OF PHOTOMIREX

Photomirex is produced through the very slow dechlorination of mirex in the presence of ultraviolet radiation, and is the chemically preferred isomer of the three possible monohydromirex isomers known to form during degradation (Kaiser 1978). Neither mirex nor photomirex is known to occur naturally in the environment (Suta 1978).

FATE AND TRANSPORT OF PHOTOMIREX

Photomirex has been found in water and sediment samples taken from Lake Ontario, and was probably present as a result of prior mirex manufacture at facilities on two tributaries to the lake, the Niagara River and the Oswego River (Holdrinet et al. 1978). Several studies have examined the fate and transport of photomirex in the water and sediments of the Lake Ontario system (Mudambi and Hassett 1988, Mudambi et al. 1992, Oliver and Niimi 1988), and low levels of photomirex have also been detected in soil samples in areas where mirex applications to control fire ants previously took place (Carlson et al. 1976).

Bioaccumulation of photomirex in lake fish and herring gulls in the Great Lakes region has been studied in detail (Braune and Norstrom 1989, Hallett et al. 1976, Hallett et al. 1977, Norstrom et al. 1978, Norstrom et al. 1980, Oliver and Niimi 1988). Photomirex has been found in several lake plants and animals, including phytoplankton, zooplankton, shrimp, oligochaete worms, sculpin, alewives, smelt, carp, eel, coho salmon, and herring gulls. Norstrom et al. (1978) estimated photomirex bioconcentration factors of 2.5×10^7 and 1.5×10^6, for herring gull eggs and coho salmon, respectively.

Air

No information on the fate and transport of photomirex in the atmosphere could be identified in the scientific literature, but photomirex is often referred to as having chemical and physical properties similar to mirex (Kaiser 1978). Because it is likely to have a very low vapor pressure (the vapor pressure of mirex is 3.0×10^{-7} mm Hg at 25°C), it is not expected to volatilize significantly to the atmosphere (IARC 1979). Any introduction into ambient air would more likely occur through contaminated dusts, which would settle rapidly. Since mirex is no longer manufactured or applied for the control of fire ants, any photomirex-contaminated dusts would most likely be limited to areas where these activities once occurred and the dusts resulting from those activities had not been environmentally dissipated.

Water

Low levels of photomirex have been detected in the waters of Lake Ontario, some of its tributaries, and its outlet. Oliver and Niimi (1988) found mean photomirex concentrations in water samples taken throughout Lake Ontario averaging 17 picograms per liter (pg/l). Mudambi et al. (1992) reported that photomirex present in Lake Ontario is produced photochemically in the water column. Photomirex concentrations varied from 1.95 to 10.4 pg/l in water samples obtained from the Oswego and Niagara Rivers, which are believed to be the most likely contributing sources of mirex and photomirex in Lake Ontario; from several locations in the lake itself; and from the St. Lawrence River, the only outlet from the lake. The authors concluded that the photomirex concentrations were most likely related to the residence time of the water in the lake; portions of the lake with longer water residence times evidently allowed more photodegradation of mirex to photomirex.

It appears that most of the photomirex in the water column exists either dissolved in the water or bound to dissolved organic matter. Yin and Hassett (1986) found that less than 10% of the mirex in water samples from the Oswego River and Lake Ontario was present in the centrifugable solids (i.e., in the sediment particles). No photomirex nor mirex was detected in centrifugable solids from water samples taken from the St. Lawrence River, although the concentrations of mirex and photomirex in the water samples (including photomirex dissolved in water and adsorbed to dissolved, noncentrifugable, organic matter) were 1.85 and 1.95 pg/l, respectively (Mudambi et al. 1992).

Soil/Sediments

Photomirex residues have been found in soils where mirex was previously applied, and in bottom and suspended sediments in Lake Ontario and the St. Lawrence River (Carlson et al. 1976, Oliver and Niimi 1988, Mudambi et al. 1992).

Carlson et al. (1976) recovered soil samples from an experimental site where mirex had been applied for fire ant control at a rate of 1 pound per acre 12 years previously. About 50% of the mirex applied was accounted for in either recovered mirex or related degradation products. The study also reported that photomirex comprised 16.1% to 19.5% of the total mirex-related residues recovered. Also, as part of this study, soil and sediment samples were recovered from the bottom of a pond into which a plane carrying "Mirex Granulated Bait 4X" (0.3% mirex) had crashed 5 years previously. The pond was dry during the spring months, exposing the mirex bait to direct sunlight. It was reported that 8.4% of the recoverable mirex and related products was in the form of photomirex.

Oliver and Niimi (1988) sampled bottom sediments and suspended sediments at several locations throughout Lake Ontario. Mean concentrations of photomirex were 3.9 and 3.7 parts per billion (ppb) (dry weight) in the bottom sediments and suspended sediments, respectively. The average ratio of photomirex to mirex in bottom sediments was 0.13.

Mudambi et al. (1992) reported that there was "very little photomirex present in sediments from the major sources of mirex to Lake Ontario ([i.e., the] Niagara and Oswego Rivers)." The concentrations of photomirex in sediments in the Niagara and Oswego Rivers were 0.03 and <0.01 ppb, respectively, whereas concentrations of photomirex in the sediments of the St. Lawrence River, the outlet from Lake Ontario, averaged 0.085 ppb. In addition, the photomirex to mirex ratio was <0.065 in the two inflows, whereas the ratio was 0.35 in the outlet river. Since mirex had not significantly degraded to photomirex in the sediments of the inflow rivers and since mirex did not degrade to photomirex in sediment samples kept in the laboratory for 4 months, the authors concluded that photomirex concentrations in the sediments of the St. Lawrence River were evidently due to the phototranformation of mirex to photomirex in the water column in the lake with subsequent adsorption of the photomirex to the sediment particles.

ENVIRONMENTAL DEGRADATION

Several studies have found that in the presence of ultraviolet radiation, the primary degradation product of mirex is photomirex (Gibson et al. 1972, Ivie et al. 1974, Carlson et al. 1976, Mudambi and Hassett 1988). Photomirex was not observed following microbial degradation of mirex (Andrade et al. 1975). Photomirex can be further degraded in sunlight to 2,8-dihydromirex (Mudambi and Hassett 1988).

Degradation of Mirex

Several researchers have shown the degradation of mirex to photomirex in the presence of sunlight. Gibson et al. (1972) reported that approximately 5% of mirex deposited on silica gel plates was converted to photomirex after being exposed to sunlight for 3 months. Ivie et al. (1974) reported that exposure of mirex, as deposits on silica gel thin-layer chromatography plates, to sunlight or ultraviolet light resulted in slow degradation to several photoproducts. Detectable levels of mirex photoproducts were observed within 3 days of exposure of the plates to sunlight. After 28 days, 11.9% of the mirex had degraded; 6.8% of the metabolites were identified as photomirex. Carlson et al. (1976) reported that 19.9% of "Mirex Granulated Bait 4X" placed in a Rayonet-type RS reactor and exposed to ultraviolet light was converted to photomirex in 19.5 hours.

Mudambi and Hassett (1988) found that mirex dissolved in distilled water at 62 nanograms per liter (ng/l) and subsequently exposed to sunlight was partially degraded to photomirex. The rate of degradation was increased with the addition of humic acids. After 6 weeks, the photomirex to mirex ratios were 3.70 and 5.43 in distilled water and distilled water with humic acids, respectively. No degradation of mirex to photomirex was observed in distilled water kept in the dark. Mudambi and Hassett (1988) also reported that the rate of mirex degradation in Lake Ontario water was greater than the degradation rate in distilled water.

After 3 weeks, the photomirex to mirex ratios were 0.7 and 0.2 for Lake Ontario water and distilled water, respectively.

Mudambi and Hassett (1988) examined the efficiency of mirex photodegradation at various wavelengths of light. The efficiency of degradation was found to be maximal at 260 nm and 265 nm in distilled water and Lake Ontario water, respectively. They found that the efficiency of degradation of mirex in the 350 to 700 nm range was lower than in the 250 to 350 nm range.

Carlson et al. (1976) reported mirex to be highly resistant to metabolic attack by soil organisms, but slow dechlorination to a monohydro derivative by anaerobic microorganisms has been observed. Andrade and Wheeler (1974) reported that mirex was not degraded in aerobic sludge but was degraded in anaerobic sludge, although the metabolites produced were not determined in that study. Andrade et al. (1975) reported in a follow-up study that the metabolite produced was not 8-monohydromirex (photomirex) but was one of the other monohydromirex metabolites.

Cripe and Livingston (1977) constructed artificial marshes to determine the significance of decomposition of mirex under natural conditions. Troughs spread with 567 grams of mirex bait (0.3% mirex) allowed entry and exit of water from the Gulf of Mexico into the artificial marshes, and were designed to allow maximum levels of sunlight to strike the bait. Photomirex concentrations in the troughs accumulated at a rate of 610 ppb per day from the 4th day to the 21st day. After 42 days, the concentration of photomirex in the bait was approximately 15 parts per million (ppm) while the concentration of mirex was approximately 1,700 ppm.

Degradation of Photomirex

The World Health Organization reports that "[t]he environmental half-life of mirex is on the order of many years, and its breakdown products are equally stable" (WHO 1984).

Alley et al. (1974) reported that photomirex is an intermediate degradation product in the formation of the dihydro photoproduct of mirex. Cripe and Livingston (1977) found photobreakdown of photomirex to either 2,8-dihydromirex or 3,8-dihydromirex in an artificial marsh system. Mudambi and Hassett (1988) found that the photolysis of mirex in water is given by the following equation:

$$\text{MIREX} \Rightarrow \text{PHOTOMIREX} \Rightarrow \text{2,8-DIHYDROMIREX}$$

In distilled water exposed to summer sunlight (Syracuse, NY), the rate of conversion of mirex to photomirex was 8.25 times greater than the rate of conversion from photomirex to 2,8-dihydromirex. In Lake Ontario water exposed to fall sunlight, the rate of conversion of

photomirex to 2,8-dihydromirex was 2.5 times greater than the rate of conversion from mirex to photomirex (Table III-1).

TABLE III-1

RATE CONSTANTS FOR THE PHOTOLYSIS OF MIREX AND PHOTOMIREX OBTAINED FROM SUNLIGHT EXPOSURE STUDIES

MEDIUM AND SUNLIGHT CONDITIONS[a]	RATE CONSTANTS[b]		
	k_1 day^{-1}	k_2 day^{-1}	k_1/k_2
Distilled water with humics/summer sunlight	0.123	0.122	1.00
Distilled water/summer sunlight	0.033	0.004	8.25
Lake Ontario water/fall sunlight	0.102	0.248	0.41
Distilled water/fall sunlight	0.019	0.125	0.15

[a] For summer sunlight conditions, samples were placed in direct sunlight in summer in Syracuse, NY. For fall sunlight conditions, samples were placed in a south-facing window in fall in Syracuse, NY. Summer sunlight conditions indicate higher percentages of available sunlight than fall sunlight conditions.

[b] The rate constant for the photolysis of mirex to photomirex is k_1. The rate constant for the photolysis of photomirex to 2,8-dihydromirex is k_2.

Source: Mudambi and Hassett 1988

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IV. HEALTH EFFECTS IN HUMANS AND ANIMALS

INTRODUCTION

The potential health effects associated with exposure to a particular substance are a function of both the dose to which an individual is exposed and the inherent toxicity of the material. Environmentally, dose is a function of the concentration of the substance in the environmental media (i.e., exposure) and the amount of the substance that reaches the target organs of concern (i.e., toxicokinetics). This section of the report addresses the potential for human exposure to photomirex, the toxicokinetics of photomirex in the body, and the potential for photomirex to cause organ damage. Each subsection is organized by route of exposure (inhalation, oral, dermal). When available, human data are discussed first, followed by animal data. In the health effects subsection, data are organized by health effect and discussed in terms of acute, intermediate, and chronic exposures. The intent of this report is to provide a comprehensive survey of the published studies related to the toxicology of photomirex.

POTENTIAL FOR HUMAN EXPOSURE

Williams et al. (1984) conducted a study of organochlorine residues in human adipose tissue samples collected during autopsies of former residents of the Great Lakes region. Mean concentrations of photomirex from the two Canadian municipalities studied were 9 ppb and 6 ppb; the highest level detected was 60 ppb.

Mes et al. (1986) conducted a study of chlorinated hydrocarbon contaminants in human breast milk. The study included 210 samples collected throughout Canada, and at least a trace of photomirex (less than 1 ppb) was found in all samples. A maximum concentration of 2 ppb was found in whole milk, and the maximum concentration found in the milkfat was 40 ppb.

TOXICOKINETICS

No studies were located which dealt with the toxicokinetics of photomirex in humans. The absorption, distribution, metabolism, and excretion of photomirex have been examined in animals following oral exposures only. The following section describes what is known about the toxicokinetics of photomirex. In general, photomirex is slowly absorbed; distributed primarily to the adipose tissue, as well as to the liver, kidney, spleen, and other organs; and excreted almost exclusively in the feces.

Absorption

Inhalation Exposure

No studies were located regarding photomirex absorption in humans or animals following inhalation exposure.

Oral Exposure

No studies were located regarding photomirex absorption in humans following oral exposure. Chu et al. (1979) found that absorption of ^{14}C-photomirex from the gastrointestinal tract in male Sprague-Dawley rats was slow. Little radioactivity could be detected in the blood 1.5 hours after oral dosing by gavage, indicating that photomirex was absorbed primarily in the small intestine rather than the stomach. The concentration of photomirex in the blood reached its peak of 8.65 ppm approximately 4 hours after administration of the dose and subsequently declined rapidly. When rats were given intravenous injections of photomirex, rapid declines of levels in the blood were also seen, indicating a redistribution of photomirex into the tissues (Chu et al. 1979).

Dermal Exposure

No studies were located regarding photomirex absorption in humans or animals following dermal exposure.

Distribution

Inhalation Exposure

No studies were located regarding photomirex distribution in humans or animals following inhalation exposure.

Oral Exposure

No studies were located regarding photomirex distribution in humans following oral exposures. However, several studies have been reported in the literature which examined the distribution of photomirex in animal tissues (Gibson et al. 1972, Chu et al. 1979, Hallett 1978, Chu et al. 1982, Fujimori et al. 1983, Villeneuve et al. 1979a, Ritter et al. 1978, Chu et al. 1981b, Sundaram et al. 1980, Villeneuve et al. 1979b, Chu et al. 1981c). Photomirex is distributed to all tissues and organs, with the highest concentrations appearing in the adipose tissue. High concentrations were also reported in the liver, kidney, and spleen. In most cases, tissue and organ residues were found to increase dose-dependently.

Gibson et al. (1972) examined the fate of the major photodecomposition product of mirex in female Sprague-Dawley rats. The mirex photoproduct used in the study was described as being either 8-monohydromirex (photomirex) or 9-monohydromirex; the authors stated that the then current methods did not permit distinction between the two isomers. Rats were given a single dose of 0.2 mg/kg of the photoproduct. Seven days after dosing, concentrations of the photoproduct in the fat averaged 1.14 ppm; concentrations of 0.06 ppm or less were detected in the brain, kidney, liver, and muscle tissue.

Chu et al. (1979) determined tissue levels of radioactivity after a single oral dose of 4.29 or 42.9 mg/kg ^{14}C-photomirex was administered to male Sprague-Dawley rats. Tissue accumulations of photomirex were dose-related. Two days after dosing, the highest concentrations were found in the fat, followed by the liver, skin, thyroid, kidneys, heart, testes, spleen, and muscle. The order of accumulation was the same in tissues examined 7, 14, 21, and 28 days after dosing (Table IV-1).

Hallett et al. (1978) studied tissue residues in male and female Wistar rats 28 days after administration of a single oral dose of photomirex at 50, 100, 150, or 200 mg/kg. The authors reported that photomirex accumulated to higher levels in adipose tissue and ovaries and was found at lower levels in liver, kidneys, spleen, heart, brain, and testes. Accumulation was dose-related, except in the fat of female rats (Table IV-2).

Fujimori et al. (1983) examined concentrations of photomirex in the organs and tissues of male ICR mice following oral administration of 10 mg/kg/day for 4 days. Mean concentrations of 26.15, 10.24, 7.80, and 1.10 ppb were observed in the liver, muscle, brain, and plasma, respectively.

Villeneuve et al. (1979a) examined concentrations of photomirex in the tissues of male Sprague-Dawley rats following dietary exposures of 0, 0.5, 5, 50, or 500 ppm photomirex for 28 days. Photomirex accumulated in a dose-dependent manner in all tissues examined; perirenal fat contained the highest concentrations. Based on estimated food intake and total body burdens, the authors determined that 10% to 20% of the photomirex ingested over the 28-day period was stored by the rat (Table IV-3).

Ritter et al. (1978) reported that male Sprague-Dawley rats exposed to 0.5, 5, 50, and 500 ppm photomirex in the diet accumulated the compound dose-dependently, with the fat containing the highest concentrations, followed by the liver, kidneys, heart, brain, and spleen.

Chu et al. (1981b) reported that male Sprague-Dawley rats fed 50 ppm photomirex for 28 days had significantly higher photomirex residue levels than controls in all examined tissues 48 weeks after completion of dosing.

TABLE IV-1

TISSUE CONCENTRATIONS OVER TIME IN MALE SPRAGUE-DAWLEY RATS AFTER A SINGLE ORAL DOSE OF 42.9 mg/kg PHOTOMIREX

TISSUE	TISSUE CONCENTRATION (ppm)[a]				
	DAY 2	DAY 7	DAY 14	DAY 21	DAY 28
Liver	65.9 ± 7.1	88.2 ± 11.1	49.4 ± 4.7	31.4 ± 6.4	21.9 ± 1.1
Kidneys	9.6 ± 1.3	8.4 ± 1.6	5.3 ± 1.3	2.8 ± 0.87	2.6 ± 0.39
Spleen	5.0 ± 0.19	4.1 ± 1.0	4.0 ± 2.9	1.8 ± 1.2	2.3 ± 0.62
Brain	11.0 ± 1.1	7.2 ± 1.3	3.1 ± 0.8	1.5 ± 0.18	1.3 ± 0.17
Bladder	16.6 ± 8.7	14.9 ± 7.4	16.4 ± 7.4	9.7 ± 6.1	7.6 ± 2.9
Skin	36.9 ± 4.0	36.1 ± 23.6	23.5 ± 4.9	28.8 ± 8.3	16.6 ± 9.5
Muscle	4.7 ± 1.1	3.9 ± 1.1	3.2 ± 1.5	3.4 ± 1.3	1.0 ± 0.5
Perirenal fat	105 ± 26	377 ± 114	284 ± 58	98.7 ± 15	85 ± 3.1
Testes	7.8 ± 1.5	4.8 ± 1.7	2.0 ± 0.42	1.0 ± 0.14	1.1 ± 0.23
Lung	19.3 ± 3.4	10.7 ± 0.76	6.0 ± 0.64	4.2 ± 2.6	3.4 ± 0.15
GI tract[b]	29.2 ± 5.3	3.8 ± 2.9	2.7 ± 0.42	0.87 ± 0.13	0.54 ± 0.14
Blood	2.07 ± 1.2	2.1 ± 0.38	--	0.54 ± 0.14	0.29 ± 0.03
Thyroid	42.9 ± 11.5	15.6 ± 5.6	7.8 ± 1.3	7.1 ± 3.2	3.4 ± 0.6
Heart	9.4 ± 1.1	6.2 ± 0.5	3.3 ± 0.15	1.5 ± 0.2	1.5 ± 0.12

[a] Mean ± standard deviation for four rats
[b] Gastrointestinal tract plus contents

Source: Chu et al. (1979)

TABLE IV-2

TISSUE RESIDUE OF PHOTOMIREX IN WISTAR RATS 28 DAYS AFTER A SINGLE ORAL DOSE

SEX	DOSE (MG/KG)	CONCENTRATION (mg/kg OF WET WEIGHT)[a] IN TISSUES						
		LIVER	HEART	BRAIN	KIDNEYS	SPLEEN	FAT	REPRODUCTIVE ORGANS
Female	200	40.8 ± 17.6	20.2 ± 12.5	16.9 ± 9.3	18.7 ± 12.0	13.0 ± 3.2	182 ± 91.0	167 ± 104
Male[b]	200	58.0	6.0	7.7	15.5	5.3	182	6.9
Female	150	30.2 ± 13.2	6.5 ± 3.0	3.5 ± 1.2	7.6 ± 1.0	7.9 ± 5.8	187 ± 114	56.1 ± 22.6
Male	150	56.4 ± 22.7	7.2 ± 2.1	5.2 ± 1.2	7.5 ± 2.6	5.4 ± 1.4	125 ± 43	4.3 ± 0.5
Female	100	12.7 ± 4.7	4.8 ± 2.0	3.4 ± 1.2	5.1 ± 1.0	4.3 ± 1.4	176 ± 59	47.7 ± 16.4
Male	100	28.0 ± 10.6	3.3 ± 1.4	2.2 ± 0.8	3.4 ± 1.6	2.2 ± 1.1	81.6 ± 17	2.1 ± 0.1
Female	50	9.7 ± 4.1	5.1 ± 1.2	4.6 ± 1.4	5.7 ± 1.2	3.7 ± 2.4	136 ± 37.9	44.4 ± 14.7
Male	50	15.2 ± 4.7	1.4 ± 0.7	1.1 ± 0.8	1.4 ± 0.7	2.5 ± 4.4	39.1 ± 16	0.8 ± 0.2

[a] Mean ± standard deviation

[b] Only one test animal was alive at the time of sacrifice

Source: Hallett et al. (1978)

TABLE IV-3

TISSUE RESIDUE CONCENTRATIONS (ppm) IN MALE SPRAGUE-DAWLEY RATS FED PHOTOMIREX FOR 28 DAYS

TISSUE	PHOTOMIREX IN DIET (ppm)		
	0.5	5.0	50
Brain	0.02 ± 0.02[a]	0.74 ± 0.25	12.5 ± 3.5
Perirenal fat	2.06 ± 0.20	22.3 ± 5.6	381 ± 196
Heart	0.08 ± 0.04	0.35 ± 0.02	12.5 ± 4.08
Liver	0.13 ± 0.04	4.6 ± 1.5	61 ± 31
Kidneys	0.12 ± 0.11	0.38 ± 0.03	7.1 ± 1.5
Spleen	0.02 ± 0.02	0.05 ± 0.02	1.6 ± 0.3

[a] Mean ± standard deviation

Source: Villeneuve et al. (1979a)

Sundaram et al. (1980) fed photomirex to female Sprague-Dawley rats in the diet at 0, 0.2, 1, 5, 25, and 125 ppm for 28 or 90 days and found that photomirex accumulated in a dose-related manner is all tissues analyzed. Highest levels were found in the perirenal fat, the liver, and the brain; photomirex was also detected in the kidneys and spleen. Accumulation in fat was found to increase with the duration of exposure, but accumulation in the liver and brain reached a steady state after 28 days.

Villeneuve et al. (1979b) examined tissue concentrations of photomirex in male Sprague-Dawley rats fed 0, 0.2, 1, 5, 25, or 125 ppm photomirex in the diet for 90 days. In a companion study, Chu et al. (1981c) examined rats after 21 months of exposure to photomirex at the same feeding levels. After 90 days, a dose-dependent accumulation of photomirex occurred in all tissues analyzed, with maximum accumulation occurring in the perirenal fat, followed by liver, kidneys, spleen, brain, and testes. After 21 months, a dose-dependent accumulation of photomirex was also observed in all tissues, with the highest levels detected in the fat, followed by the liver, testes, brain, spleen, heart, and kidneys.

Tissue distribution of photomirex following intravenous injection is similar to that observed following ingestion. Chu et al. (1982) examined the distribution of photomirex in squirrel monkeys 16, 34, and 52 weeks after a single intravenous injection of 20 mg/kg. Three animals were examined; sacrifices were at 16, 34, or 52 weeks. The highest concentrations of photomirex were found in the fat, followed by skin, pancreas, adrenals, liver, and nerves. The fat, skin, liver, and muscle also served as storage sites due to their large masses. Redistribution of radioactivity was reported to occur between 16 and 34 weeks. At 16 weeks, the muscle and skeleton contained 32% of the administered dose and the skin and subcutaneous fat contained 12%; at 34 weeks, the muscle and skeleton contained only 4.1% of the administered dose and the fat contained 71%.

Dermal Exposure

No studies were located regarding photomirex distribution in humans or animals following dermal exposure.

Metabolism

No studies were located regarding photomirex metabolism in humans. Several studies conducted to evaluate the potential metabolism of photomirex in animals reported that the compound is evidently not metabolized. Chu et al. (1979) detected no metabolites of photomirex in the tissues or feces of male Sprague-Dawley rats administered single doses of 4.29 or 42.9 mg/kg photomirex by gavage. Chu et al. (1982) examined the metabolism of ^{14}C-labelled photomirex in squirrel monkeys at 16, 34, or 52 weeks after a single intravenous injection of 20 mg/kg. No metabolites of photomirex were detected when the radioactive materials in the fat, skin, liver, and feces were extracted.

Excretion

Inhalation Exposure

No studies were located regarding photomirex excretion in humans or animals following inhalation exposure.

Oral Exposure

No studies were located regarding photomirex excretion in humans following oral exposure. Several studies conducted in animals have shown that photomirex is excreted primarily in the feces (Gibson et al. 1972, Chu et al. 1979, Chu et al. 1982).

Gibson et al. (1972) examined the excretion of the major photodecomposition product of orally administered mirex in female Sprague-Dawley rats. The mirex photoproduct used in the study was described as being either 8-monohydromirex (photomirex) or 9-monohydromirex; the authors stated that analytical methods current at the time did not permit distinction between the two isomers. Rats orally dosed with 0.2 mg/kg of the photoproduct excreted 18.4% in their feces within 7 days, with 80% of that amount excreted within the first 24 hours. Only 0.13% of the dose was excreted in the urine within the 7 days.

Chu et al. (1979) examined the excretion of photomirex in male Sprague-Dawley rats following a single oral dose by gavage of 4.29 or 42.9 mg/kg photomirex in corn oil. Similar patterns of excretion were observed at both levels; photomirex was eliminated almost exclusively in the feces. During the first 3 days following dosing, 38% to 42% of the administered photomirex was eliminated unchanged in the feces. Within 28 days, 51% to 55% of the dose was eliminated. Less than 0.09% of the original dose was excreted in the urine in the first 24 hours.

Chu et al. (1982) examined the excretion of photomirex in squirrel monkeys for 16, 34, or 52 weeks after a single intravenous injection of 20 mg/kg. Cumulative excretion of photomirex in feces ranged from 5.9% to 10.3% of the total dose; excretion in urine was insignificant.

Dermal Exposure

No studies were located regarding photomirex excretion in humans or animals following dermal exposure.

DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

In order to fully consider the potential for photomirex to cause adverse health effects in humans, the following sections, organized according to exposure route (inhalation, oral, dermal) and then by health effect (death, systemic, immunological, neurological, developmental, reproductive, genotoxic, and carcinogenic effects), are intended to present a comprehensive survey of the published studies related to the toxicology of photomirex.

Inhalation Exposure

No studies were located which reported health effects in humans or animals following inhalation exposure to photomirex.

Oral Exposure

Death

No poisoning case reports or epidemiology data were located which reported lethality in humans following oral exposure to photomirex. Studies in rats and mice in which deaths were observed are summarized below.

Photomirex appears to be more acutely toxic to the male rat than to the female rat. Hallett et al. (1978) reported that single doses of 200 mg/kg photomirex administered to Wistar rats by gavage produced 40% mortality in females and 80% mortality in males within 28 days. The authors of the study concluded that the 200-mg/kg dose likely approached the LD_{50} for photomirex. The LD_{50} is the dose of a substance that can be expected to cause death in 50% of the animals.

Sundaram et al. (1980) reported 40% and 20% mortality in female Sprague-Dawley rats exposed to 125 ppm in the diet for 28 and 90 days, respectively. Cyanosis (a bluish discoloration from excess reduced hemoglobin in the blood) and irritability were noted prior to death. Chu et al. (1981c) reported that 9 out of 10 male Sprague-Dawley rats exposed to 125 ppm photomirex in the diet died between the 3rd and 19th week of a 21-month chronic toxicity study; the final rat died after 55 weeks. Clinical signs of toxicity included hypoactivity, irritability, and cyanosis of the hind limbs. Villeneuve et al. (1979b) reported that 4 out of 10 male Sprague-Dawley rats fed 125 ppm photomirex in the diet died within 8 weeks; clinical symptoms prior to death included weight loss, hyperactivity, cyanosis of the hind limbs, and irritability.

Villeneuve et al. (1979a) reported that all male Sprague-Dawley rats exposed to photomirex at 500 ppm in the diet died within 7 days; this dose was equivalent to 133 mg/kg/day. Clinical symptoms prior to death included irritability, tremors, hypoactivity, and a mild

cyanosis in the hind limbs. Ritter et al. (1978) reported that 9 out of 10 male Sprague-Dawley rats exposed to photomirex at 500 ppm in the diet died within 28 days.

Several acute toxicity studies conducted in mice have also been reported. Fujimori et al. (1980) reported that male ICR mice exposed to photomirex at 25 and 50 ppm in their diet died within 15 and 7 days, respectively. The authors estimated an LT_{50} of 225 to 250 mg/kg; the LT_{50} is the cumulative daily dose which kills 50% of the test animals. Fujimori et al. (1983) orally dosed male ICR mice with 10 mg/kg/day photomirex until death of all animals occurred; the LT_{50} value for photomirex determined in this study was 265 mg/kg. This study determined lower LT_{50} values for mirex and kepone, following dosing with 10 mg/kg/day, of 132 and 254 mg/kg, respectively. However, when mice were dosed with 25 or 50 mg/kg/day photomirex, mirex, or kepone, the LT_{50} values for all three compounds were similar.

Systemic Effects

No studies were located which reported systemic effects in humans following exposure to photomirex. The available information on the potential health effects of photomirex is based on toxicological studies on experimental animals that were dosed orally in most cases; in several studies, dosing occurred intravenously.

The reviewed studies indicate that photomirex causes a variety of toxic responses which affect the liver, thyroid, and testes most severely. No studies were located which reported cardiovascular, respiratory, or musculoskeletal effects following oral exposure to photomirex in animals.

Hematological effects. Hematological parameters in rats exposed to photomirex were generally within the ranges of normal values. However, some hematological effects were seen at higher dosing levels.

Villeneuve et al. (1979a) found hemoglobin concentrations, hematocrit values, erythrocyte counts, total and differential counts of leukocytes, mean corpuscular volumes, and mean corpuscular hemoglobin counts within normal ranges in male Sprague-Dawley rats fed 0, 0.5, 5, or 50 ppm photomirex in their diets for 28 days.

Chu et al. (1981b) reported normal hematological parameters in male Sprague-Dawley rats 0, 12, 24, and 48 weeks after receiving 0, 0.05, 0.5, 5, or 50 ppm photomirex in their feed for 28 days. Hematological parameters examined included hemoglobin concentrations, hematocrit values, erythrocyte counts, total and differential counts of leukocytes, mean corpuscular volumes, mean corpuscular hemoglobin concentrations, mean corpuscular hemoglobins, and cytological evaluations of bone marrow smears.

Ritter et al. (1978) reported a significant increase in white blood cell counts in male Sprague-Dawley rats fed 50 ppm photomirex in the diet for 28 days.

Sundaram et al. (1980) reported normal hematological parameters in female Sprague-Dawley rats receiving 0, 0.2, 1, 5, 25, or 125 ppm photomirex in their feed for 28 days and in female Sprague-Dawley rats receiving 0, 0.2, 1, 5, or 25 ppm photomirex in their feed for 90 days. Total hemoglobin contents, erythrocyte counts, and mean corpuscular hemoglobin contents were all significantly decreased in rats fed photomirex at 125 ppm in the diet for 90 days. Mean corpuscular volumes were significantly elevated in rats fed photomirex at 125 ppm in the diet for 90 days. Hematological parameters examined included hemoglobin contents, hematocrit values, erythrocyte counts, total and differential counts of leukocytes, mean corpuscular volumes, mean corpuscular hemoglobin concentrations, and mean corpuscular hemoglobins.

Villeneuve et al. (1979b) found hematological parameters within normal ranges in weanling male Sprague-Dawley rats fed 0, 0.2, 1, 5, 25, or 125 ppm photomirex in their diets for 90 days. Hematological parameters examined included hemoglobin concentrations, hematocrit values, erythrocyte counts, total and differential counts of leukocytes, mean corpuscular volumes, mean corpuscular hemoglobin concentrations, and mean corpuscular hemoglobins. Although statistical analysis revealed no statistical differences in leukocyte differential counts, there was a trend towards greater numbers of neutrophils (polynuclear leukocytes) with increasing photomirex concentrations. Also, there were incidences of hypersegmented neutrophils in all treatment groups which increased in a dose-related manner. In a companion study, Chu et al. (1981c) found that hematological parameters were also not altered after exposure to photomirex at the same feeding levels for 21 months. Hematological parameters examined at this time included hemoglobin concentrations, hematocrit values, total and differential counts of leukocytes, mean corpuscular volumes, mean corpuscular hemoglobin concentrations, mean corpuscular hemoglobins, and cytological evaluations of bone marrow.

Chu et al. (1981a) reported normal hematological parameters in female Sprague-Dawley rats fed 2.5, 5, 10, 20, or 40 ppm photomirex in their diets for 91 days prior to mating, 15 days during mating, and throughout gestation and lactation. Hematological parameters examined included hemoglobin concentrations, packed cell volumes, erythrocyte counts, total and differential counts of leukocytes, mean corpuscular volumes, mean corpuscular hemoglobin concentrations, and cytological evaluations of bone marrow smears.

Hepatic effects. There are a number of published reports on the induction of adverse hepatic effects from orally administered photomirex in rats and mice. These studies reported liver weight increases, histological lesions, and alterations in hepatic enzyme activities as the most common effects.

Chambers and Trevathan (1983) reported hepatic effects 2 days after single oral doses of 100 mg/kg photomirex in corn oil were administered to female Sprague-Dawley rats. Liver weights and hepatic microsomal protein and ascorbic acid concentrations were significantly increased over controls (increased by 38%, 30%, and 131%, respectively). Total hepatic protein and hepatic glutathione levels were not significantly altered from controls. The cytochrome P-450 activity of hepatic microsomes was induced 4-fold by photomirex. Specific activities of NADPH-cytochrome *c* reductase, aminopyrine *N*-demethylase (APDM), and *p*-nitroanisole *O*-demethylase were increased significantly over control levels.

Fujimori et al. (1983) reported that male ICR mice fed 10 mg/kg photomirex daily for 4 consecutive days had liver weights significantly greater than those of controls. Cytochrome P-450, cytochrome *c* reductase, and NADPH dehydrogenase activities, and ligand interactions with aniline and hexobarbital were also significantly increased over controls.

Mehendale et al. (1979) examined several parameters associated with the hepatic mixed-function oxidase system in male Sprague-Dawley rats fed 50 ppm photomirex in the diet for 15 days. Liver-to-body weight ratios were increased by 67.5% over controls. The activities of NADPH oxidase, cytochrome P-450, NADPH-cytochrome *c* reductase, APDM, and hepatic aniline hydroxylase (AH) were all significantly increased over controls at this feeding level.

Curtis et al. (1979) examined biliary excretion functions following exposure of male Sprague-Dawley rats to 50 or 150 ppm photomirex in the diet for 15 days. Impaired hepatobiliary function, as measured by the excretion of two anionic model compounds, polar metabolites of imipramine (PMIMP) and phenolphthalein glucuronide (PG), was observed. Significant dose-dependent depression in Mg^{2+}-ATPase activity was reported in both treatment groups. Serum glutamic oxalacetic transaminase (SGOT) activities were not affected.

Strik et al. (1980) reported a significant increase in liver weight in female U-strain rats fed 100 ppm photomirex in the diet for 27 days. Histological examinations of the livers revealed moderate injuries, characterized by pericentral cytoplasmic enlargements with peripheralized cytoplasmic basophilia and overall reduction in cytoplasmic density. In addition, cells exhibited nuclear vesiculations and anisokaryosis (inequalities in the size of cell nuclei).

Villeneuve et al. (1979a) examined the hepatic effects of photomirex in male Sprague-Dawley rats fed 0, 0.5, 5, 50, or 500 ppm photomirex in their diets for 28 days. Mortality of all test animals in the highest dose group occurred within 7 days. Liver weights and liver-to-body weight ratios were significantly increased in the 5- and 50-ppm groups. Serum sorbitol dehydrogenase (SDH) and hepatic AH activities were significantly increased at the 5- and 50-ppm feeding levels; increases in the activities

of these enzymes were dose-dependent. Histopathological lesions appeared in the livers at all test levels and were characterized by progressive cytoplasmic enlargement, anisokaryosis, hyperchromicity (excessive pigmentation suggesting degeneration of the cell nuclei), and fatty infiltration.

Chu et al. (1981b) examined the reversibility of toxic effects in male Sprague-Dawley rats fed diets containing 0, 0.05, 0.5, 5, or 50 ppm photomirex for 28 days; histological examinations were conducted at the conclusion of dosing, and also at 12, 24, and 48 weeks after photomirex-free feed was introduced. Congested livers were observed in the 0.5, 5, and 50-ppm groups at the conclusion of dosing; the congestion was still observed at 48 weeks. Liver-to-body weight ratios were significantly elevated in the 5- and 50-ppm groups at the conclusion of dosing, and in the 50-ppm group at 12 and 24 weeks. SGOT activities were not different from controls at the conclusion of dosing. However, significant depressions in SGOT activities were observed in the 0.5-,5-, and 50-ppm groups at 12 weeks; levels were unchanged from controls at 24 weeks. SDH activities were near levels seen in controls at the conclusion of dosing, but were significantly elevated in the 50-ppm group at 12 weeks. Although AH and APDM activities were normal at the conclusion of dosing and at 12 weeks, both levels were significantly elevated at 24 weeks in the 50-ppm group; APDM activities were still increased at 48 weeks. Lactic dehydrogenase (LDH) activities were decreased in the 50-ppm group at the conclusion of dosing, but returned to normal within 12 weeks. Pathological changes observed in livers at the conclusion of dosing included cytoplasmic vacuolation, reduction in aggregated basophilia, nuclear anisokaryosis, and hyperchromicity of hepatocytes. At higher doses, pericentral fatty vacuolation was also seen. These changes were not seen in the two lower dose groups at 48 weeks, but were still present in the livers of the two higher dose groups at that examination. This study also examined livers of rats fed equivalent amounts of mirex; less severe histopathological changes in the livers of rats fed mirex were observed, even at 48 weeks.

Yarbrough et al. (1981) evaluated hepatic effects in male Sprague-Dawley rats fed 0, 0.5, 5, 50, or 75 ppm photomirex in their diets for 28 days. Significant increases in liver-to-body weight ratios were noted in the 5-, 50-, and 75-ppm treatment groups. Liver SDH levels were significantly elevated at all treatment levels. Microsomal protein levels, cytochrome P-450 activities, and AH activities were all significantly increased in the 5-, 50-, and 75-ppm feeding groups. APDM activities were significantly increased in the 0.05- and 75-ppm feeding groups. Serum β-glucuronidase activities were significantly reduced in the 5-, 50, and 75-ppm groups. During histological examinations, there were signs of mild and subtle cytoplasmic alterations in the liver tissues of animals fed 0.5 ppm photomirex, although this change could not be consistently identified. Cytoplasmic changes were identified in all livers of animals in the 5-, 50-, and 75-ppm groups. This study also examined hepatic effects in rats fed mirex in the diet at the same levels, and found no significant differences in rat responses to the two compounds.

Ritter et al. (1978) examined hepatic effects of photomirex in male Sprague-Dawley rats fed 0, 0.5, 5, 50, and 500 ppm photomirex in the diet for 28 days. Animals fed 0.5, 5, and 50 ppm photomirex had significantly increased liver weights. Microscopic examinations of the livers showed mid-zonal balloonings of the hepatocytes accompanied by regressive changes in hepatocellular nuclei.

Sundaram et al. (1980) fed photomirex to female Sprague-Dawley rats in the diet at 0, 0.2, 1, 5, 25, and 125 ppm for 28 or 90 days. Other animals in the study were fed mirex or kepone. Liver weights and liver-to-body weight ratios were increased in rats fed photomirex at 25 or 125 ppm for 28 or 90 days. SDH activities were significantly increased in the 25- and 125-ppm groups fed photomirex in the diet for 90 days. Histological lesions (vacuolation, ballooning of cells, reduction of basophilia, fatty vacuolation) were observed in rats fed photomirex at 1 ppm or greater in the diet for 28 or 90 days; the effects were dose-dependent. When compared with data on male rats, female rats were found to be less susceptible to liver effects than the male rats exposed to photomirex. Mirex and kepone had similar effects on the liver but the changes were mild and less severe at comparable doses, leading the investigators to conclude that "photomirex was approximately five times more toxic than mirex in terms of liver histology."

Villeneuve et al. (1979b) examined the hepatic effects of photomirex in male Sprague-Dawley rats fed 0, 0.2, 1, 5, 25, or 125 ppm photomirex in their diets for 90 days. Dose-dependent statistically significant increases in liver weights and hepatic AH activities were observed at the 5-ppm and greater dose levels. Dose-related histological changes were observed in livers starting at the lowest dose level.

Chu et al. (1981a) conducted a reproductive study in which male and female Sprague-Dawley rats were fed diets containing 0, 2.5, 5, 10, 20, or 40 ppm photomirex for 13 weeks prior to mating and for 15 days during mating; female rats continued on this diet throughout gestation and lactation. Enlarged livers were noted in the adult females in the highest dose group. Hepatic APDM activities were significantly increased in adult females and in their offspring in all treatment groups.

In a companion study to the Villeneuve et al. (1979b) study, Chu et al. (1981c) examined hepatic effects of photomirex in male Sprague-Dawley rats fed 0, 0.2, 1, 5, 25, and 125 ppm in the diet for 21 months. All rats in the 125-ppm group died during the study; livers from these animals were enlarged and mottled, and histological examinations indicated severe liver damage characterized by cytoplasmic enlargement of hepatocytes, nuclear hyperchromicity, anisokaryosis, necrosis (cell death), and fatty infiltration. Significantly increased liver weights and liver-to-body weight ratios occurred in groups dosed at 5 ppm and higher. Significant increases in hepatic microsomal AH and APDM activities occurred in the groups fed 1 and 25 ppm photomirex. Activities of serum SDH in the 25-ppm group, SGOT in the 0.2-ppm group, and LDH in the 1-ppm group were all significantly elevated.

Treatment-related histological lesions were found in the livers of all rats in all photomirex treatment groups and became progressively more severe as dosages increased.

Thyroid effects. There are a number of published reports on the induction of histological changes and lesions in the thyroids of rats orally administered photomirex.

Chu et al. (1981c) reported thyroid lesions in male Sprague-Dawley rats that received 125 ppm photomirex in their diet until death (3 to 55 weeks). The thyroid lesions consisted of generalized reductions in follicle sizes and an angular collapse of follicles with increased epithelial heights and reduced and irregular colloid densities.

Villeneuve et al. (1979a) examined thyroid effects of photomirex in male Sprague-Dawley rats fed 0, 0.5, 5, 50, or 500 ppm photomirex in their diets for 28 days. All test animals in the highest dose group died within 7 days. Histopathological lesions appeared in the thyroid/parathyroids of the 50- and 500-ppm groups, and consisted of progressive reductions in colloid densities and increases in follicular atrophies.

Yarbrough et al. (1981) examined thyroid effects in male Sprague-Dawley rats fed 0, 0.5, 5, 50, or 75 ppm photomirex in their diets for 28 days. Serum triidothyronine (T_3) levels were significantly different from controls in the 75-ppm group and serum thyroxine (T_4) levels were significantly reduced in the 50- and 75-ppm groups. Photomirex induced histological changes which included hypertrophy and elongation of the lining epithelial cells, reduction and/or depletion of colloid density, follicular atrophy, and a tendency to produce focal papillary formations (nipple-shaped projections) within the lining epithelial cells. These changes were more pronounced in rats treated at 50 and 75 ppm. This study also examined thyroid effects in rats fed mirex in the diet and found no significant differences in rat response to the two compounds.

Chu et al. (1981b) examined the thyroid glands and thyroid functions of male Sprague-Dawley rats following exposure to 0.05, 0.5, 5, or 50 ppm photomirex in the diet for a period of 28 days. Examinations were conducted at the conclusion of dosing and after 12, 24, and 48 weeks on a photomirex-free diet. Serum T_3 and T_4 levels were not significantly different from controls in any group at the conclusion of dosing and after 12, 24, and 48 weeks on a photomirex-free diet. Changes in the thyroid consisted of reductions in colloid densities, angular collapses of follicles, and increases in epithelial heights with nuclear vesiculations. After 48 weeks on a photomirex-free diet, only mild changes in the thyroids at reduced frequencies remained. Singh et al. (1985) examined the thyroids of rats in the same study for histological changes 48 weeks after conclusion of dosing. Doses of 0.05 and 0.5 ppm produced cytoplasmic vacuolations in the principal follicular cells. Doses of 5 ppm produced a mild dilation of rough-surfaced endoplasmic reticulum, reductions in the

number of apical vesicles, and deformed lysomal bodies in the columnar follicular cells. The follicular cells of the thyroids of rats fed 50 ppm photomirex had more severe alterations, including marked dilations of the rough-surfaced endoplasmic reticulum profiles.

In a later study, Singh et al. (1982) examined the thyroid glands of male Sprague-Dawley rats for 18 months following exposures to 0.05, 0.5, 5, or 50 ppm photomirex in their diets for a period of 28 days. Doses of 0.05 and 0.5 ppm resulted in changes in the follicular cell shapes from cuboidal (in controls) to columnar and marked increases in the number of secondary lysosomes. Doses of 5 ppm resulted in similar changes in the shapes of follicular cells; however, follicular cells in some segments of the gland contained an increased number of lysosomes, while cells in other segments contained fewer. At 50 ppm, the shapes of the follicular cells were also elongated and the numbers of secondary lysosomal elements were increased; additionally, some of the cells contained enlarged colloid droplets. The study reported that the ultrastructure alterations seen in the thyroid glands at all dose levels persisted for at least 18 months. This study also examined thyroid effects in rats fed mirex in the diet at the same dose levels. No alterations in thyroid ultrastructure in rats fed 0.05, 0.5, and 5 ppm mirex were noted; the thyroids of rats fed 50 ppm mirex exhibited elongated follicular cells and increases in the number of secondary lysosomes.

Sundaram et al. (1980) examined thyroid effects of photomirex in female Sprague-Dawley rats fed 0, 0.2, 1, 5, 25, or 125 ppm photomirex in the diet for 28 and 90 days. Thyroids of rats fed 25 ppm for 90 days exhibited mild reductions in colloid volumes and densities, reductions in follicle size, angular collapses of larger peripheral follicles, and increases in the heights of epithelial cells. Changes were more pronounced in animals fed photomirex at 125 ppm. The study reported a no-effect level of 1 ppm for the 28-day study and 0.2 ppm for the 90-day study, suggesting an additive effect of exposure level and exposure duration. Serum T_3 and T_4 levels were unchanged in all groups fed photomirex for 28 days and 90 days, with the exception of the 125-ppm group fed photomirex for 90 days. In this group, serum T_3 levels were unchanged, while serum T_4 levels were significantly reduced. Thyroid effects produced by exposures to mirex appeared to be less severe.

Villeneuve et al. (1979b) examined thyroid effects of photomirex in male Sprague-Dawley rats fed 0, 0.2, 1, 5, 25, or 125 ppm photomirex in the diet for 90 days. Serum T_3 levels were not affected at any dose; serum T_4 levels were significantly decreased in the 5-ppm and 125-ppm groups. Dose-related histological abnormalities were observed in the thyroid starting at the lowest dose level and included reductions in colloid volumes with collapses of larger peripheral follicles and increases in the height of the epithelium. In a companion study, Chu et al. (1981c) examined the thyroids of rats fed photomirex at the same dietary levels for

21 months. Changes in thyroids from rats in the 0.2- and 1-ppm groups included irregular reductions in follicular sizes and colloid densities and increases in epithelial heights. Changes in thyroids from rats in the 5- and 25-ppm groups included a generalized reduction in follicular sizes and colloid densities and increased epithelial heights over the heights seen in the 0.2- and 1-ppm groups.

Renal effects. Photomirex has been reported to affect the kidneys of rats at dietary levels of 50 ppm and higher. Hallett et al. (1978) reported mottled and congested kidneys after single oral doses of 50, 100, 150, and 200 mg/kg photomirex in male and female Wistar rats. Villeneuve et al. (1979a) reported a significant reduction in kidney weights (but not kidney-to-body weight ratios) in male Sprague-Dawley rats fed 50 ppm photomirex in their diets for 28 days. Ritter et al. (1978) reported significant increases in kidney weights in male Sprague-Dawley rats fed 50 ppm photomirex in their diets for 28 days. Chu et al. (1981b) examined the reversibility of toxic effects in male Sprague-Dawley rats fed diets containing 0, 0.05, 0.5, 5, or 50 ppm photomirex for 28 days; examinations occurred 0, 12, 24, and 48 weeks after photomirex-free feed was introduced. Kidney-to-body weight ratios were still significantly elevated in the 50-ppm group after 12 and 48 weeks of photomirex-free feed.

Gastrointestinal effects. Fujimori et al. (1983) reported mild diarrhea and hemorrhagic intestines in male ICR mice fed 10, 25, or 50 mg/kg photomirex daily until death.

Other systemic effects. Several studies reported changes in body weights, food intakes, and organ weights in rats given photomirex in the diet (Ritter et al. 1978, Villeneuve et al. 1979a, Sundaram et al. 1980, Villeneuve et al. 1979b, Chu et al. 1981b). Ritter et al. (1978) reported significant increases in food intakes, body weight gains, and heart and spleen weight gains in male Sprague-Dawley rats fed 50 ppm photomirex in the diet for 28 days. Villeneuve et al. (1979a) reported significant reductions in body weight gains, food intakes, and heart and kidney weights in male Sprague-Dawley rats fed 50 ppm photomirex in their diets for 28 days. Significant reductions in spleen weights and significant increases in liver weights and liver-to-body weight ratios were observed in the 5- and 50-ppm groups. Sundaram et al. (1980) reported decreased body weight gains and food intakes in female Sprague-Dawley rats fed photomirex in the diet at 125 ppm for 28 days, but not in rats fed the same diets for 90 days. Villeneuve et al. (1979b) reported significant depressions in food intakes and body weight gains in male rats after 90-day exposure to photomirex in the diet at 125 ppm.

Chu et al. (1981b) undertook a study to determine the reversibility of changes seen in previous studies of male rats (Villeneuve et al. 1979a, 1979b). Male Sprague-Dawley rats were fed diets containing 0, 0.05, 0.5, 5, or 50 ppm photomirex for 28 days and then were placed on photomirex-free feed; examinations occurred at the conclusion of

dosing, and at 12, 24, and 48 weeks after photomirex-free feed was introduced. Rats in the 50-ppm group showed significantly reduced food intakes and body weight gains at the conclusion of dosing and at 12 weeks, but did not differ significantly from controls in food uptakes and body weight gains at 24 or 48 weeks.

Some studies reported changes in biochemical parameters in mice and rats which were not reported in the sections above (Fujimori et al. 1983, Yarbrough et al. 1981, Chu et al. 1981b). Fujimori et al. (1983) examined the effects of photomirex on blood glucose, lactate, free fatty acids, and brain, liver, and muscle glycogen in male ICR mice following oral administration of 25 mg/kg/day for 4 days. Blood glucose levels (after a 24-hour fast) and lactate levels (after a 6-hour fast) were significantly decreased. The mobilization of free fatty acids into the blood after a 6-hour fast was not altered significantly from controls. Brain and muscle glycogen levels did not differ significantly from controls; liver glycogen levels were significantly decreased.

Yarbrough et al. (1981) found that blood glucoses and total proteins were significantly reduced in male Sprague-Dawley rats fed 75 ppm photomirex in the diet for 28 days. Chu et al. (1981b) fed male Sprague-Dawley rats diets containing 0, 0.05, 0.5, 5, or 50 ppm photomirex for 28 days and found that serum inorganic phosphorus levels were significantly elevated at the conclusion of dosing in the 0.5- and 50-ppm groups. Serum inorganic phosphorus levels were still significantly elevated in the 50-ppm group 12 weeks after dosing was completed.

Immunological Effects

No studies were located which reported immunological effects in humans and animals following oral exposure to photomirex.

Neurological Effects

No studies were located which reported neurological effects in humans following oral exposure to photomirex. The most common neurological effect found in animals exposed to high doses of photomirex was tremor (Villeneuve et al. 1979a, Chu et al. 1981a). Villeneuve et al. (1979a) reported that male Sprague-Dawley rats exposed to photomirex at 500 ppm in their diet developed clinical symptoms including irritability, tremor, hypoactivity, and a mild cyanosis in the hind limbs, and died within 7 days. Chu et al. (1981a) reported clinical signs of toxicity such as hypoactivity, irritability, and muscle tremor in male and female Sprague-Dawley rats fed 40 ppm photomirex in their diets for 106 days.

Developmental Effects

No studies were located which reported developmental effects in humans following oral exposure to photomirex. In a teratogenicity study in New Zealand white rabbits, females

were administered doses of 0, 5, or 10 mg/kg photomirex on days 6 through 18 of gestation (Villeneuve et al. 1979c). None of the animals showed any signs of toxicity and no teratogenic effects were seen in the offspring. A significant reduction in the mean fetal weight in the 10 mg/kg-dose group was noted. Tissue analysis at term showed the highest concentrations of photomirex in the mothers were in the fat, followed by the liver, kidneys, spleen, heart, brain, and blood. In the fetus, the highest concentrations of photomirex were found in the heart, followed by the liver, brain, and blood.

Chu et al. (1981a) reported the formation of cataracts in the eyes of some offspring of male and female Sprague-Dawley rats fed 10 or 20 ppm photomirex in their diets for 91 days prior to and for 15 days during mating. Female rats were kept on this diet throughout gestation and lactation.

Reproductive Effects

No studies were located which reported reproductive effects in humans following oral exposure to photomirex. Ingestion of photomirex has been demonstrated to cause testicular damage in rats and mice.

Effects on male reproduction system. Photomirex has been reported to affect spermatogenesis (sperm production) and the ultrastructural histology of the testes in rats at high doses (Chu et al. 1981b, Villeneuve et al. 1979a, Yarbrough et al. 1981). Photomirex has also been reported to cause sperm abnormalities in mice (Hugenholtz et al. 1984, Hugenholtz and Douglas 1986).

Chu et al. (1981b) observed cessation of spermatogenesis and complete aspermia in the epididymides of male Sprague-Dawley rats fed diets containing 50 ppm photomirex for 28 days. These changes were not apparent after rats were placed on a photomirex-free diet for 12 weeks, although loss of spermatogonia and cessation of spermatogenesis were found after 24 weeks on a photomirex-free diet. After 48 weeks on a photomirex-free diet, unilateral testicular atrophy with loss of spermatogenic cells was noted.

Yarbrough et al. (1981) reported a significant reduction in sperm counts in male Sprague-Dawley rats fed 5 and 50 ppm photomirex in the diet for 28 days, but not in rats fed 75 ppm photomirex. Villeneuve et al. (1979a) reported histopathological lesions in the testes of male Sprague-Dawley rats fed 50 ppm photomirex in their diets for 28 days. Lesions included complete cessation of spermatogenesis and marked tubular degeneration. Villeneuve et al. (1979b) reported significant decreases in testicular SDH activities in male Sprague-Dawley rats fed 125 ppm photomirex in their diets for 90 days.

Photomirex is reported to cause sperm abnormalities in mice. Hugenholtz et al. (1984) treated male B6C3F1 mice with photomirex for 5 consecutive days at "total doses up to 80% of the LD_{50}." They observed increases in levels of sperm abnormalities but found no effects

on testes weights or sperm counts. Hugenholtz and Douglas (1986) examined sperm abnormalities in B6C3F1 mice and Sprague-Dawley rats every 2 weeks for 12 weeks after 5 consecutive daily intraperitoneal injections with photomirex at 1.8, 9, and 18 mg/kg/day for the mice, and at 3.3, 16.5, and 33 mg/kg/day for the rats. Treatments increased sperm abnormalities in mice, reaching maximum levels 6 to 12 weeks after treatment. Body weights, testes weights, and sperm numbers were unaffected by the treatments.

Effects on female reproduction system. The female rat is suspected of being much less susceptible to injury of the reproductive organs by photomirex than is the male rat (Sundaram et al. 1980). Histological changes in reproductive organs may be associated with decreases in body weight gains since both effects were observed in female Sprague-Dawley rats exposed to 25 or 125 ppm photomirex for 28 days, but not for 90 days (Sundaram et al. 1980). Hallett et al. (1978) reported hemorrhagic ovaries in female Wistar rats given a single oral dose of 100, 150, or 200 mg/kg photomirex.

Chu et al. (1981a) conducted a reproductive study to determine the effects of daily photomirex intake on female rats and their offspring. Male and female Sprague-Dawley rats were fed diets containing 0, 2.5, 5, 10, 20, or 40 ppm photomirex for 13 weeks prior to mating and for 15 days during mating; female rats continued on this diet throughout gestation and lactation. Females fed 40 ppm showed a significant decrease in weight gain but not in food consumption. The numbers of females showing sperm in vaginal smears were decreased in all treatment groups (significance not determined). Litter sizes were decreased in all treatment groups, although the decreases were not significant in the 2.5-ppm group. Gestational, 4-day, and 21-day survival indices in pups were only affected in the highest-dose group. Three weeks after birth of the pups, both mothers and surviving offspring were sacrificed. Enlarged livers and minor biochemical changes in blood sera were noted in the adult females in the highest dose group.

Genotoxic Effects

No studies were located which reported genotoxic effects of photomirex in humans. Hugenholtz et al. (1984) found no effect of photomirex on micronucleus frequency in peripheral blood samples collected from male B6C3F1 mice treated with photomirex for 5 consecutive days at "total doses up to 80% of the LD_{50}". Hallett et al. (1978) tested the mutagenicity of photomirex using the standard Ames bacterial assay including a microsomal activation mixture. The results of the test indicated that photomirex was not mutagenic.

Cancer

No studies were located which reported potentially carcinogenic effects of photomirex in humans following oral exposures to photomirex. Chu et al. (1981c) examined tissues of male Sprague-Dawley rats fed 0, 0.2, 1, 5, or 25 ppm photomirex in the diet for 21 months. Thyroid adenomas were observed in 4 of 10 animals in the 25-ppm group and in 1 of 10

control animals; none of the animals in the other exposure groups exhibited thyroid adenomas.

Dermal Exposure

No studies were located which reported health effects of photomirex in humans or animals following dermal exposure to photomirex.

LEVELS IN HUMAN TISSUE AND FLUIDS ASSOCIATED WITH EFFECTS

Because no information is available concerning health effects in humans exposed to photomirex, it is not possible to determine levels in human tissues and fluids associated with toxicity.

LEVELS IN THE ENVIRONMENT ASSOCIATED WITH LEVELS IN HUMAN TISSUES AND/OR HEALTH EFFECTS

Insufficient information is available concerning the relationships between ambient levels of photomirex, body burdens of photomirex, and toxicities to determine environmental levels of photomirex potentially associated with human toxicities.

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V. ENVIRONMENTAL AND REGULATORY CRITERIA

An integral part of the human health risk assessment process is the identification of levels of toxic agents that may pose potential risks to humans via specific exposure pathways. In its present form, this process involves the derivation of a reference dose (RfD), or reference concentration (RfC) for inhalation exposures, which is an estimate of the daily exposure that is unlikely to pose an appreciable risk of adverse effects in a human population, and/or a cancer slope factor (CSF), which is an estimate of the risk per unit dose associated with exposure to a potentially carcinogenic substance.

The EPA Risk Assessment Guidance for Superfund, Human Health Evaluation Manual (EPA 1989) recommends procedures for determining RfDs and CSFs for potentially toxic substances. The process begins with a search of the various EPA reference sources to determine whether the Agency has calculated and verified an RfD or CSF. When such criteria are not available, EPA recommends the calculation of RfDs and CSFs from data available in the open literature.

A thorough search revealed that EPA has not developed either an RfD or a CSF for photomirex. Therefore, the suitability of available toxicological data for calculating these criteria were evaluated. Inherent in this process was the evaluation of studies in the literature for scientific method, adequacy of data, sources of interference, and suitability of application. Studies that were directly related to human toxicity were preferred over other studies. Studies utilizing common test species ranked second in utility. Studies involving uncommon test species or uncontrolled field observations were considered least valuable.

CURRENT REGULATORY STATUS

No current EPA regulatory standards concerning photomirex were identified.

CARCINOGENICITY EVALUATION

Only one identified study provided information on the potential carcinogenicity of photomirex. Chu et al. (1981c) examined tissues of male Sprague-Dawley rats fed 0, 0.2, 1, 5, or 25 ppm photomirex in the diet for 21 months. Although many tissues were examined, only the thyroid glands were thought by the study investigators to possibly contain tumors related to the administration of photomirex. Thyroid adenomas were observed in 4 of 10 animals in the 25-ppm group and in 1 of 10 control animals; none of the animals in the other exposure groups exhibited thyroid adenomas. Despite the fact that subtle ultrastructural changes in the thyroids were observed in some rats at doses as low as 0.2 ppm, insufficient data are available to establish an association between the histological abnormalities and any increased cancer risk. In fact, ultrastructural changes of the thyroid were observed in all

animals in the 25-ppm group, yet thyroid adenomas were only observed in 4 of the 10 animals. Furthermore, histological changes in the thyroid gland occur naturally as animals age, are thought to be reversible, and are of uncertain clinical relevance (see section on the evaluation of noncarcinogenic effects, p. V-2). No other studies which reported the occurrence of ultrastructural changes in the thyroids of rats exposed to photomirex reported similar evidence of thyroid tumors (Chu et al. 1981b, Singh et al. 1982, Singh et al. 1985, Villeneuve et al. 1979b, Sundaram et al. 1980).

Chu et al. (1981c) concluded that the thyroid tumors in the animals receiving 25 ppm photomirex "might" be considered to be treatment related. However, no statistical analyses were provided to indicate that the incidences of these lesions were significantly elevated. Reported background incidences of thyroid tumors in the rat are dependent on a number of factors, including the strain of rat, histopathological procedures, diagnostic criteria, and the age of the animal at autopsy. Nevertheless, it should be noted that some researchers have concluded that spontaneous thyroid tumors are relatively common in some strains, and that a background incidence of thyroid adenomas as high as 44% in male Sprague-Dawley rats has been reported (IARC 1976, Thompson and Hunt 1963). This value is comparable with that reported by Chu et al. (1981c) for rats exposed to photomirex at 25 ppm.

The Chu et al. (1981c) study also suffers from a number of inadequacies that further limit its usefulness for evaluating the potential carcinogenicity of photomirex: (1) it provides results for only one sex and strain of rat with no corroborative data from other studies; (2) very small numbers of animals were employed in the experimental groups in comparison with typical carcinogenicity studies; (3) "spontaneous" deaths occurred prior to completion of the study in all groups (including control animals) except in the 25-ppm photomirex group; (4) details on the histopathological procedures used were not provided; and (5) only benign thyroid tumors were reported.

Overall, the Chu et al. (1981c) report is an inadequate study of the potential carcinogenic activity of photomirex as defined by the National Toxicology Program. Because of major qualitative and quantitative limitations, it cannot be interpreted as valid for showing either the presence or absence of carcinogenic activity. Consideration of the available data on the potential carcinogenicity of photomirex indicates that this compound is most appropriately classified in the Environmental Protection Agency's Weight-of-Evidence Group D, Not Classifiable as to Human Carcinogenicity. This classification is generally used for agents with inadequate human and animal evidence of carcinogenicity or for which no data are available.

EVALUATION OF NONCARCINOGENIC EFFECTS

The EPA RfD/RfC Work Group has not developed a chronic oral reference dose (RfD) for photomirex that has been verified for inclusion in EPA's Integrated Risk Information System (IRIS) database. Therefore, an RfD for photomirex has been calculated below in accordance

with recommendations contained in EPA's Risk Assessment Guidance for Superfund, Human Health Evaluation Manual (EPA 1989). In the absence of human data, animal studies were reviewed to identify the Lowest-Observed-Adverse-Effect-Levels (LOAELs) and No-Observed-Adverse-Effect-Levels (NOAELs) following oral exposures to photomirex. Insufficient human data were available from which to develop an oral reference dose; therefore, animal data were reviewed to identify the critical toxic effect of photomirex used to derive the RfD. The studies considered are summarized in Table V-1.

Selection of Critical Data

The EPA guidelines for developing an RfD require identification of the critical toxic effect, which is the effect characterized by the lowest-observed-adverse effect level (LOAEL) and to then identify the highest dose tested that did not result in the critical adverse effect (NOAEL). In the current analysis of photomirex, consideration was given to studies which reported critical toxic effects at the lowest levels for each relevant endpoint. NOAELs and LOAELs reported as ppm photomirex in feed were converted to equivalent doses in mg/kg/day, using mean daily feed consumption and animal body weight data provided in the respective studies. Where animal body weights and food consumption data were not reported, conversions were carried out using standard values recommended by EPA (1986). The body weight estimates for rats and mice were 0.03 kg and 0.35 kg, respectively. Food consumption rates were estimated as a fraction of total body weight by multiplying the body weight by 0.05 for rats and 0.13 for mice. Figure V-1 graphically depicts the NOAELs and LOAELs identified from a survey of the experimental studies of photomirex toxicity in animals.

Thyroid Effects

A review of subchronic and chronic oral exposures to photomirex (Table V-1) revealed that the lowest dose reported to cause an adverse effect following oral exposure to photomirex is 0.05 ppm (0.0025 to 0.005 mg/kg/day, depending on food intake and body weight parameters) administered in the diets of male Sprague-Dawley rats for 28 days (Chu et al. 1981b, Singh et al. 1982, Singh et al. 1985). Histological examinations of the thyroids of these animals revealed ultrastructural changes including elongations of the follicular cells and increases in the numbers of secondary lysosomes. Because these changes were observed at the lowest dose tested, a NOAEL could not be identified.

Other studies reported similar subtle histological changes in the thyroid. Villeneuve et al. (1979b) observed histological changes in the thyroids of male Sprague-Dawley rats fed 0.2 ppm photomirex (0.02 mg/kg/day) in the diet for 13 weeks. This was the lowest dose administered in the study. Sundaram et al. (1980) observed histological changes in the thyroids of female Sprague-Dawley rats fed 1 ppm photomirex (0.126 mg/kg/day) in the diet for 90 days. The NOAEL for this study was 0.2 ppm or 0.027 mg/kg/day. Changes in the

TABLE V-1

EXPERIMENTAL STUDIES OF ORAL EXPOSURE TO PHOTOMIREX

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	ENDPOINT	NOAEL[a] (mg/kg/day)	LOAEL[b]		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
1	Mouse/ICR	Gavage	4 days	Liver weight; liver enzyme activity	---	10[c]	---	Fujimori et al. 1983
2	Mouse/ICR	Gavage	6 days	Body weight	10[c]	25[c]	---	Fujimori et al. 1980
3	Rat/ Sprague-Dawley	Diet	15 days	Liver weight; liver enzyme activity (AH,APDM)	---	3.33[c] (50 ppm)	---	Mehendale et al. 1979
4	Rat/ Sprague-Dawley	Diet	15 days	Liver-to-body weight ratio; hepatobiliary function	---	3.37[c] (50 ppm)	---	Curtis et al. 1979
5	Mouse/ICR	Diet	17 days	Food consumption	---	1.3[c] (10 mg/kg)	---	Fujimori et al. 1980
6	Rat/U-strain	Diet	27 days	Liver weight	---	5[d] (100 ppm)	---	Strik et al. 1980
7	Rat/ Sprague-Dawley	Diet	28 days[e]	Thyroid and liver histologic effects	---	0.005[c] (0.05 ppm)	---	Singh et al. 1985; Chu et al. 1981b

TABLE V-1

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO PHOTOMIREX
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	ENDPOINT	NOAEL[a] (mg/kg/day)	LOAEL[b]		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
8	Rat/ Sprague-Dawley	Diet	28 days[e]	Liver enzyme activity (SGOT)	0.005[c] (0.05 ppm)	0.05[c] (0.5 ppm)	---	Chu et al. 1981b
9	Rat/ Sprague-Dawley	Diet	28 days[e]	Liver weight	0.05[c] (0.5 ppm)	0.5[c] (5 ppm)	---	Chu et al. 1981b
10	Rat/ Sprague-Dawley	Diet	28 days	Liver weight	---	0.025[c] (0.5 ppm)	---	Ritter et al. 1978
11	Rat/ Sprague-Dawley	Diet	28 days[f]	Thyroid histological effects	---	0.0025[c] (0.05 ppm)	---	Singh et al. 1982
12	Rat/ Sprague-Dawley	Diet	28 days	Liver histological effects	0.027[d] (0.2 ppm)	0.126[d] (1 ppm)	---	Sundaram et al. 1980
13	Rat/ Sprague-Dawley	Diet	28 days	Thyroid histological effects	0.126[d] (1 ppm)	0.636[d] (5 ppm)	---	Sundaram et al. 1980
14	Rat/ Sprague-Dawley	Diet	28 days	Food consumption; body weight gain	0.68[c] (5 ppm)	6.61[c] (50 ppm)	---	Villeneuve et al. 1979a
15	Rat/ Sprague-Dawley	Diet	28 days	Liver histological effects	---	0.08[c] (0.5 ppm)	---	Villeneuve et al. 1979a

TABLE V-1

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO PHOTOMIREX
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	ENDPOINT	NOAEL[a] (mg/kg/day)	LOAEL[b]		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
16	Rat/ Sprague-Dawley	Diet	28 days	Reproductive toxicity (cessation of spermatogenesis)	0.68[c] (5 ppm)	---	6.61[c] (50 ppm)	Villeneuve et al. 1979a
17	Rat/ Sprague-Dawley	Diet	28 days	Reproductive toxicity (sperm count)	0.028[c] (0.5 ppm)	---	0.28[c] (5 ppm)	Yarbrough et al. 1981
18	Rat/ Sprague-Dawley	Diet	28 days	Liver weight	0.028[c] (0.5 ppm)	0.28[c] (5 ppm)	---	Yarbrough et al. 1981
19	Rat/ Sprague-Dawley	Diet	28 days	Thyroid histological effects	0.28[c] (5 ppm)	2.8[c] (50 ppm)	---	Yarbrough et al. 1981
20	Rat/ Sprague-Dawley	Diet	28 days	Liver enzyme activity (SDH, APDM)	---	0.028[c] (0.5 ppm)	---	Yarbrough et al. 1981
21	Rat/ Sprague-Dawley	Diet	90 days	Thyroid histological effects	0.027[d] (0.2 ppm)	0.126[d] (1 ppm)	---	Sundaram et al. 1980
22	Rat/ Sprague-Dawley	Diet	90 days	Liver histological effects	0.027[d] (0.2 ppm)	0.126[d] (1 ppm)	---	Sundaram et al. 1980
23	Rat/ Sprague-Dawley	Diet	90 days	Hematological parameters	3.11[d] (25 ppm)	16.70[d] (125 ppm)	---	Sundaram et al. 1980

TABLE V-1

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO PHOTOMIREX
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	ENDPOINT	NOAEL[a] (mg/kg/day)	LOAEL[b]		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
24	Rat/ Sprague-Dawley	Diet	13 weeks[g]	Body weight gain; food consumption	2.24[c] (25 ppm)	12.2[c] (125 ppm)	---	Villeneuve et al. 1979b
25	Rat/ Sprague-Dawley	Diet	13 weeks[g]	Liver histological effects	---	0.02[c] (0.2 ppm)	---	Villeneuve et al. 1979b
26	Rat/ Sprague-Dawley	Diet	13 weeks[g]	Reproductive toxicity (testicular enzyme activity, SDH)	2.24[c] (25 ppm)	---	12.2[c] (125 ppm)	Villeneuve et al. 1979b
27	Rat/ Sprague-Dawley	Diet	13 weeks[g]	Thyroid histological effects	---	0.02[c] (0.2 ppm)	---	Villeneuve et al. 1979b
28	Rat/ Sprague-Dawley	Diet	91 days prior to mating, 15 days during mating, throughout gestation and lactation	Body weight	1 (20 ppm)	2 (40 ppm)	---	Chu et al. 1981a
29	Rat/ Sprague-Dawley	Diet	91 days prior to mating, 15 days during mating, throughout gestation and lactation	Liver enzyme activity (hepatic APDM)	---	0.125[d] (2.5 ppm)	---	Chu et al. 1981a

TABLE V-1

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO PHOTOMIREX
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	ENDPOINT	NOAEL[a] (mg/kg/day)	LOAEL[b]		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
30	Rat/ Sprague-Dawley	Diet	91 days prior to mating, 15 days during mating, throughout gestation and lactation	Liver lesions	0.25 (5 ppm)	0.5 (10 ppm)	---	Chu et al. 1981a
31	Rat/ Sprague-Dawley	Diet	21 months[g]	Liver weight	0.06[c] (1 ppm)	0.34[c] (5 ppm)	---	Chu et al. 1981c
32	Rat/ Sprague-Dawley	Diet	21 months[g]	Liver enzyme activity (SGOT)	---	0.013[c] (0.2 ppm)	---	Chu et al. 1981c
33	Rat/ Sprague-Dawley	Diet	91 days prior to mating, 15 days during mating, throughout gestation and lactation	Developmental toxicity (cataracts in offspring)	0.25 (5 ppm)	---	0.5 (10 ppm)	Chu et al. 1981a
34	Rat/ Sprague-Dawley	Diet	91 days prior to mating, 15 days during mating	Reproductive toxicity (litter size)	0.125[d] (2.5 ppm)	---	0.25[d] (5 ppm)	Chu et al. 1981a

TABLE V-1

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO PHOTOMIREX
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	ENDPOINT	NOAEL[a] (mg/kg/day)	LOAEL[b]		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
35	Rabbit/ New Zealand	Gavage	GD 6-18[h]	Developmental toxicity (fetal weight)	5	---	10	Villeneuve et al. 1979c

[a] The no-observed-adverse-effect-level (NOAEL) is the highest exposure level at which no harmful effects were seen in the organ system studied.

[b] The lowest-observed-adverse-effect-level (LOAEL) is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects in order to identify the levels of exposure at which adverse health effects first appear and the reversibility or the gradation of the effects with increasing dose.

[c] Dose given to male rats only.

[d] Dose given to female rats only.

[e] Singh et al. 1985 and Chu et al. 1981b report results from the same study with different observation times. In this study, the reversibility of the effects of photomirex was studied by dosing the animals for 28 days and observing them at intervals of 12, 24, and 48 weeks on a clean diet. Hematological parameters were normal throughout exposure and recovery. Liver-to-body weight ratios were different only during the exposure period. Food consumptions and body weight gains were different through the exposure period and after 12 weeks on a clean diet. Reproductive effects were seen during the exposure period but not after 12 weeks on a clean diet. However, these reproductive effects were seen once again after 24 weeks and unilateral atrophy with loss of spermatogenic cells in the males was noted after 48 weeks. Changes in the thyroid were only mild after 48 weeks on a clean diet. Congested livers and inflated kidney-to-body weight ratios persisted after the 48 weeks recovery period.

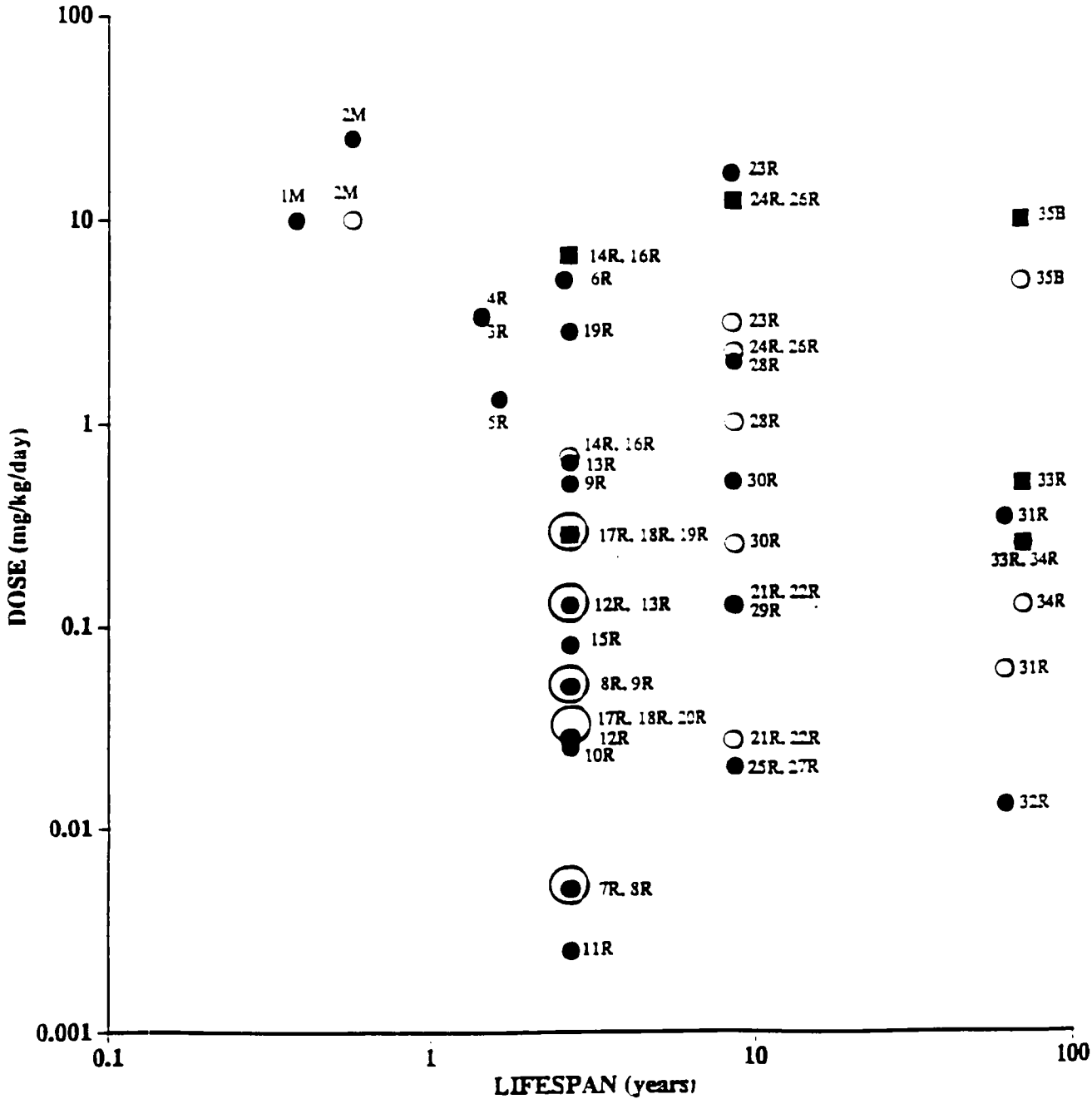
[f] These results were observed following a 28-day exposure period and an 18-month recovery period.

[g] Villeneuve et al. 1979b and Chu et al. 1981c are companion studies with different observation times.

[h] In this study, animals were administered photomirex on gestation days (GD) 6-18. Uterine contents of the does were examined on GD 30.

FIGURE V-1

EFFECT-DOSE-DURATION FOR PHOTOMIREX ANIMAL STUDIES



The data points plotted above correspond with the individual studies summarized in Table V-1. Numbers refer to figure key and R, M, and B refer to rat, mouse, and rabbit respectively. Effect levels are indicated by the following symbols: ■ = LOAEL (serious effects); ● = LOAEL (less serious effects); ○ = NOAEL. It should be noted that, in some cases, LOAELs for less serious effects may actually be more appropriately characterized as NOAELs and that some NOAELs may be more appropriately characterized as NOELs. Dose durations are divided by the appropriate species lifespan to yield a fraction which, when multiplied by 70 years (the assumed average human lifespan), gives the corresponding position on the X-axis.

thyroids of rats examined included reduction in colloid volume with collapse of larger peripheral follicles and increased height of follicular cells.

The NOAELs and LOAELs identified from these studies were considered inappropriate for developing the RfD for several reasons, including the questionable use of ultrastructural change as an appropriate toxicity endpoint, the fact that changes in the thyroid gland ultrastructure are a part of the natural aging process, the examination of a single sex of rats in each of the studies, the inability to determine the statistical significance of ultrastructural changes, the reversal of ultrastructural changes over a recovery observation period, and the subjectivity and sampling bias inherent in examination of electron microscopic tissue samples.

It is not clear whether the appearance of thyroid ultrastructural changes of the types noted in these studies are evidence of a toxic response of the thyroid or are simply changes that may not be related to photomirex administration and which may not affect thyroid function. Measurement of thyroxine (T_4) and triiodothyronine (T_3) levels in the blood generally provides an indication of thyroid function. These two hormones are the major secretions of the thyroid and affect the metabolic rate of the body. They are synthesized in the follicular colloid and the follicular epithelial cells of the thyroid. Several of the studies in which histological changes were observed in the thyroids of rats exposed to photomirex also measured serum T_3 and T_4 levels (Chu et al. 1981b, Singh et al. 1985, Yarbrough et al. 1981, Sundaram et al. 1980, Villeneuve et al. 1979b). These studies found that, in general, T_3 and T_4 levels were unaffected by photomirex exposures at the low doses at which minor ultrastructural changes in the thyroid were observed. Serum T_4 levels were not altered below dietary levels of photomirex of 50 ppm; serum T_3 levels were not affected below dietary levels of photomirex of 75 ppm. Serum T_3 and T_4 levels in rats fed dietary levels of 0.05 to 50 ppm photomirex for 28 days (Chu et al. 1981b, Singh et al. 1985) or 0.2 to 125 ppm photomirex for 90 days (Villeneuve et al. 1979b) were not different from controls. Yarbrough et al. (1978) fed rats dietary levels of 0.5 to 75 ppm photomirex for 28 days and found that serum T_3 levels were only significantly different from controls in the 75-ppm group, and serum T_4 levels were significantly reduced in the 50- and 75-ppm groups. In 90-day studies, in which rats were fed 0.2 to 125 ppm photomirex, serum T_3 levels were not affected (Sundaram et al. 1980, Villeneuve et al. 1979b). However, serum T_4 levels were affected in the 125-ppm groups in both studies and in the 5-ppm group in the Villeneuve et al. (1979b) study; however, Villeneuve et al. (1979b) did not find a dose-related relationship between photomirex dose and serum T_4 level.

Histological changes in the thyroid gland occur naturally as the animal ages. Rats used in the studies conducted by Singh et al. (1982, 1985) and Chu et al. (1981b) were weanlings (about 3 weeks old) at the beginnings of the studies, which ran for 48 weeks and 18 months. Many of the changes seen in the normal aging rat thyroid are similar to those ultrastructural changes seen in the studies of photomirex. Fujita et al. (1980) examined the thyroids of male CF_1 mice ranging in age from 7 days to 24 months. They found that the variability of follicle diameters increased and the mean diameters of the follicles became larger with

advancing age, due to fusions of adjacent follicles. They also found that the volumetric densities of lysosomes in the cytoplasm of epithelial cells increased with age. Garner and Bernick (1975) examined the thyroids of both male and female Holtzman and Wistar rats from birth to 26 months old. They found that the follicular cells were cuboidal in shape until the animals were about 10 months in age; between 10 and 15 months, the follicular cells became columnar in shape and contained intracellular colloid droplets.

It is also not possible to determine the physiological significance of the ultrastructural changes seen by Singh et al. (1982, 1985), Chu et al. (1981b), Villeneuve et al. (1979b), and Sundaram et al. (1980) based on the information provided in these studies. The Singh et al. (1982, 1985) studies did not report how many animals actually exhibited the thyroid changes described in the results. Chu et al. (1981b) reported that 3 out of 9 thyroids contained changes at the conclusion of dosing. Villeneuve et al. (1979b) reported that 3 out of 9 thyroids had changes, whereas Sundaram et al. (1980) reported that 4 out of 5 had changes. However, in all cases, no statistical analyses were completed to determine if the numbers of alterations represented statistically significant increases from controls, or whether the increases were independent of thyroid changes naturally occurring in rats. The studies also indicated that only some unknown percentage of observed follicles were altered but did not provide any quantitative assessments of this percentage. Also, the studies reported altered thyroid follicles in the control groups as well.

Furthermore, the ultrastructural changes appearing in the thyroids were reported by Chu et al. (1981b) to be completely reversible in the 0.05-ppm feeding group. While 3 out of 9 thyroids contained lesions when examined ultrastructurally after conclusion of dosing, thyroids from groups examined 12, 24, and 48 weeks after the completion of dosing were claimed to exhibit no lesions. Lesions were also reversible at all other levels of photomirex exposure.

For the reasons described above, ultrastructural changes in the thyroids of rats exposed to low levels of photomirex in the diet are not appropriate endpoints to use for photomirex systemic toxicity. While the functioning of the thyroid and its ability to produce hormones, as measured by T_3 and T_4 levels in the blood, may be appropriate toxic endpoints, changes in these hormone levels occur at much higher exposures than changes in the thyroid ultrastructure, but below levels at which other toxic effects have been reported.

Liver Effects

Several studies reported changes in the ultrastructure, enzyme activities, and weights of the livers of rats following exposures to photomirex. The lowest dose reported to cause ultrastructural changes in the liver following oral exposure to photomirex is 0.05 ppm (0.005 mg/kg/day) administered in the diet of male Sprague-Dawley rats for 28 days (Chu et al. 1981b). Histological examinations of the livers revealed ultrastructural changes including cytoplasmic vacuolation, aggregated basophilia, nuclear anisokaryosis, and hyperchromicity. Because these changes were observed at the lowest dose tested, a NOAEL could not be

identified. This study also measured liver enzyme activities, including serum glutamic oxalacetic transaminase (SGOT), serum lactic dehydrogenase (LDH), serum sorbitol dehydrogenase (SDH), hepatic microsomal aniline hydroxylase (AH), and hepatic microsomal aminopyrine *N*-demethylase (APDM). There were no significant changes in the levels of any of these enzymes at the 0.05-ppm feeding level at the conclusion of dosing, or at 12, 24, or 48 weeks after dosing was completed.

Villeneuve et al. (1979b) observed histological changes in the livers of male Sprague-Dawley rats fed 0.2 ppm photomirex (0.02 mg/kg/day) in the diet for 13 weeks. The authors reported that changes at this level consisted of a distinct lobular pattern. This was the lowest dose administered in the study and, therefore, a NOAEL was not identified. No significant changes in any measured liver enzyme activities or significant increases in liver weight were observed below the 5-ppm feeding level. At this level, liver weight and AH activity were significantly increased. Significant increases in serum SDH activities were observed at 125 ppm, but no changes were observed in LDH and SGOT levels at any feeding level.

Villeneuve et al. (1979a) observed histopathologic changes in livers of male Sprague-Dawley rats feed 0.5 ppm photomirex in the diet for 28 days. At this level, changes were characterized by mild midzonal cytoplasmic enlargements around the hepatic vein. This was the lowest dose administered. Significant increases in liver weight, serum SDH activities, and hepatic AH activities were not observed below the 5-ppm feeding level. Sundaram et al. (1980) observed histological changes in the livers of female Sprague-Dawley rats fed 1 ppm photomirex (0.126 mg/kg/day) in the diet for 28 and for 90 days. The NOAEL determined in this study was 0.2 ppm (0.027 mg/kg/day). Changes in the livers of rats examined from both feeding durations included vacuolations of central cells in a perinuclear manner with increased hyperchromicity. No other hepatic effects were observed in these studies at the 1-ppm level. Significant increases in liver weights and liver-to-body weight ratios were observed at the 25-ppm feeding level. No changes in liver enzyme activities, including AH, SGOT, and serum SDH, were observed at any feeding level in rats dosed for 28 days. Significant changes in SGOT and SDH levels were observed at the 125-ppm and 25-ppm feeding levels, respectively (AH was not measured) in rats dosed for 90 days.

The LOAELs and NOAEL identified in these studies based on ultrastructural changes in the liver were considered inappropriate for developing the RfD for several reasons including the questionable use of ultrastructural changes as appropriate toxicity endpoints, the examination of a single sex of rats in each of the studies, the inability to determine the statistical significance of ultrastructural changes, the reversal of ultrastructural changes over a recovery observation period, and the subjectivity and sampling bias inherent in examination of electron microscopic tissue samples.

In all of the studies in which liver ultrastructural changes were noted, liver enzyme activities were also measured. As described above, no changes in the activities of these enzymes occurred at the feeding levels at which ultrastructural changes were first observed, indicating that normal liver function had not been affected. Many of the studies included the

measurement of SGOT activities, an aminotransferase enzyme used as a standard to provide an indication of liver injury. Chu et al. (1981b) found that SGOT levels were significantly elevated in the 0.5-, 5-, and 50-ppm feeding groups 12 weeks after the conclusion of dosing, but not at the conclusion of dosing; however, 24 weeks after the conclusion of dosing, SGOT levels were no longer elevated. No changes in SGOT activities were observed at any feeding level (up to and including 125 ppm) by Villeneuve et al. (1979b) or in the 28-day study by Sundaram et al. (1980). In the 90-day study by Sundaram et al. (1980), SGOT activities were significantly altered only at the 125-ppm feeding level. SGOT activities were not measured by Villeneuve et al. (1979a). The inconsistent changes in SGOT activities, combined with the lack of correlation with doses or recovery periods, makes the enzyme changes inappropriate for use in establishment of an RfD for photomirex.

It is not possible to determine the significance of the ultrastructural changes reported based on information provided in the studies. None of the studies found lesions in all livers examined at the LOAELs. No statistical analyses were completed to determine if the histological changes found in some of the examined livers represented statistically significant increases from controls or whether any increase was independent of liver changes naturally occurring in rats. Furthermore, the ultrastructural changes appearing in the livers were reported by Chu et al. (1981b) to be completely reversible in the 0.05-ppm feeding group. While 5 out of 9 livers contained lesions when examined at the conclusion of dosing, livers from groups examined 12, 24, and 48 weeks after the completion of dosing were reported to exhibit 2, 1, and 0 lesions, respectively. Lesions were also reversible at all other levels of photomirex exposure.

Several studies reported changes in liver enzyme activities as a result of oral exposures to photomirex. Chu et al. (1981c) reported the lowest dose sufficient to cause a significant change in liver enzyme activities was 0.2 ppm photomirex (0.013 mg/kg/day), a dose that produced significant increases in SGOT activities in male Sprague-Dawley rats after 21 months of dietary exposure. This LOAEL was considered inappropriate for developing the RfD because changes in SGOT activities were not dose-dependent, only a single sex of rat was examined, and the significance of rat liver enzyme activities in determination of toxicity is unknown. While supposedly significant increases in SGOT levels were observed in the group fed 0.2 ppm photomirex in the diet, similar increases were not observed in rats in this study fed 1, 5, or 25 ppm photomirex. Additionally, all other liver enzyme activities measured in this study, including serum LDH and SDH, and hepatic microsomal AH and APDM, exhibited non-dose-dependent changes in activities.

Yarbrough et al. (1981) reported significant increases in liver SDH and APDM activities in male Sprague-Dawley rats fed 0.5 ppm photomirex (0.028 mg/kg/day) in diets for 28 days. This was the lowest dose administered in the study. This LOAEL was considered inappropriate for developing the RfD because elevations in liver SDH and APDM activities were not dose-dependent, other liver enzyme activities were not affected, and only one sex of rat was examined. Serum SDH and AH activities, also measured in this study, were not significantly different from controls at any feeding levels (0.5 to 125 ppm photomirex).

Chu et al. (1981b) reported a significant depression in SGOT activities in male Sprague-Dawley rats fed 0.5 ppm photomirex (0.028 mg/kg/day) in the diet for 28 days. Although SGOT activities were not different from controls at the conclusion of dosing, activities were significantly inhibited 12 weeks later. The NOAEL determined from this study was 0.05 ppm (0.0028 mg/kg/day). This NOAEL was considered inappropriate for developing the RfD because reductions in SGOT activities were transitory and were not apparent at the conclusion of dosing or at 24 and 48 weeks after the conclusion of dosing. Also, in other studies examining photomirex exposures and SGOT activities, no significant dose-related deviations of SGOT activities from controls were observed at photomirex feeding levels up to 125 ppm and for dosing periods as long as 21 months (Chu et al. 1981a, Chu et al. 1981c, Villeneuve et al. 1979b, Sundaram et al. 1980).

Chu et al. (1981a) observed significant increases in hepatic microsomal APDM activities in female Sprague-Dawley rats fed 2.5 ppm photomirex (0.125 mg/kg/day) in the diet for 13 weeks prior to mating, for 15 days during mating, and throughout gestation and lactation. This was the lowest dose administered in the study. This LOAEL was considered inappropriate for developing the RfD for several reasons, including that fact that no changes were observed in this study in any other liver enzyme activities, changes in APDM activity were not noted in any other studies in which the effects of photomirex exposure were studied, and the significance of rat liver enzyme activity levels in determination of toxicity is unknown. Chu et al. (1981a) also reported no significant changes in activities of other liver enzymes measured at any level in the study; enzymes activities studied included SGOT, serum LDH and SDH, and hepatic microsomal AH. Chu et al. (1981b) did not find significant increases in APDM in male Sprague-Dawley rats fed 0.05 to 50 ppm photomirex in the diet for 28 days, except at the highest feeding level. Chu et al. (1981c) (feeding levels of 0.2 to 25 ppm photomirex) and Yarbrough et al. (1981) (feeding levels 0.1 to 125 ppm photomirex) both found some incidences of increased APDM activity in male Sprague-Dawley rats, but these incidences were sporadic and not dose related.

Changes in liver weights in rats were also observed following oral exposures to photomirex. Ritter et al. (1978) observed significant increases in liver weights in male Sprague-Dawley rats fed 0.5 ppm photomirex (0.025 mg/kg/day) in the diet for 28 days. The liver weight increases do not appear to have been dose-dependent, although there was insufficient information provided in the study to make an absolute determination. The lowest dose level at which significant liver weight changes were seen was the lowest dose administered in the study; therefore, a NOAEL was not identified. This LOAEL was considered inappropriate for developing the RfD because increases in the liver weights did not appear to be dose-dependent, only one sex of rat was examined in the study, and insufficient information was available to evaluate the significance of the changes observed and the quality of the study. Several other studies examined liver weights and liver-to-body weight ratios and found significant increases in these parameters occurred at feeding levels of 5 ppm in rats (Chu et al. 1981b, Yarbrough et al. 1981, Villeneuve et al. 1979b, Chu et al. 1981c). All of these studies also employed a 0.5- or 1-ppm feeding level in which significant liver weight changes were not observed.

Because of the reasons given, it is not appropriate to use the LOAELs and NOAELs described above, based on hepatic effects, to calculate an RfD for photomirex. The ultrastructural changes in the livers of rats exposed to low levels of photomirex in the diet are not appropriate endpoints to use for systemic toxicities because measured liver enzyme levels did not change correspondingly at these low levels. In fact, dose-related statistically significant changes in enzyme activities were not noted in any of the studies. More importantly, the reviewed studies did not reach a consensus on the effects of photomirex on hepatic enzyme activities. While significant changes in liver weights may be an appropriate endpoint, the weight-of-evidence suggests that liver weights were not affected at dietary levels below 5 ppm in rats.

Reproductive Effects

Several studies reported reproductive effects in animals following ingestion of photomirex. These effects included hemorrhagic ovaries, cessation of spermatogenesis, and reduction in sperm counts in rats and sperm abnormalities in mice (Hallett et al. 1978, Chu et al. 1981b, Villeneuve et al. 1979a, Yarbrough et al. 1981, Hugenholtz et al. 1984, Hugenholtz and Douglas 1986). However, reductions in litter size occurred at lower dietary levels than any of these effects. Chu et al. (1981a) observed significant reductions in litter sizes when male and female Sprague-Dawley rats were fed 5 ppm photomirex (0.25 mg/kg/day) in the diet for 13 weeks prior to mating and for 15 days during mating; female rats continued on this diet throughout gestation and lactation. A NOAEL for photomirex of 2.5 ppm (0.125 mg/kg/day) was identified from this study which was the highest dose at which no significant change in litter size was observed.

Calculation of the RfD

The oral RfD was calculated according to standard procedures described in EPA guidance (EPA 1986). Studies which reported the lowest LOAELs for each relevant toxic endpoint were considered. Based on the analysis above, the study chosen to use as a basis for the RfD was the reproductive study performed by Chu et al. (1981a). In this study, reproductive effects (litter size reductions) were seen at a LOAEL of 0.25 mg/kg/day, with a NOAEL of 0.125 mg/kg/day. Effects seen in the liver and the thyroid at lower levels were not considered appropriate for use in development of the RfD; the effects reported were either histological changes not considered physiologically significant or non-dose-dependent changes in liver weights or liver enzyme activities (an extensive discussion of the analysis is provided on the preceding pages).

According to EPA guidance for calculating an RfD (EPA 1986), the NOAEL, or in the absence of an appropriate NOAEL, the LOAEL, is modified by application of generally order-of-magnitude uncertainty factors that reflect various types of data sets used to estimate RfDs. An uncertainty factor of 100 was applied to the NOAEL for photomirex of

0.125 mg/kg/day identified for the Chu et al. (1981a) reproductive study in rats to account for interspecies extrapolation (x10) and to account for sensitive individuals in the population (x10). The resulting RfD for photomirex is 0.00125 mg/kg/day.

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APPENDIX F

A Review of Kepone

A REVIEW OF KEPONE

December 2, 1992

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I. INTRODUCTION

Kepone is an environmentally persistent pesticide product that was manufactured in the United States from the 1950s until 1975. The use of this compound was discontinued in 1976 when all United States Environmental Protection Agency (EPA) registrations for products containing kepone were canceled due to concerns about its potential carcinogenicity. From the 1950s to 1975, approximately 1,600,000 kg of kepone were manufactured in the United States (Epstein 1978, IARC 1979, WHO 1984) and distributed in a number of products used predominately outside of the country.

Kepone can still be found today in certain environmental settings, despite the fact that it is no longer manufactured or used. Historically, kepone was used for a variety of pest control applications. Accidental or non-use releases into the environment, most of which occurred many years ago, have been the principal sources of environmental kepone residues in the United States. Such introductions included the incidental release of kepone and kepone dusts from manufacturing, distribution, transportation, and storage.

Perhaps the most publicized environmental release of kepone occurred in the vicinity of Hopewell, Virginia, where Life Science Products Corporation released kepone into the municipal sewage collection system and allowed fugitive dusts to contaminate the surrounding environment. These releases took place from 1974 to 1975 and resulted in widespread contamination of the James River and sections of the Chesapeake Bay.

The purpose of this report is to identify the published literature characterizing the potential human health risks associated with exposure to kepone. The organization of this report is designed to present both the hazard-related data and the context in which those data have specific meaning.

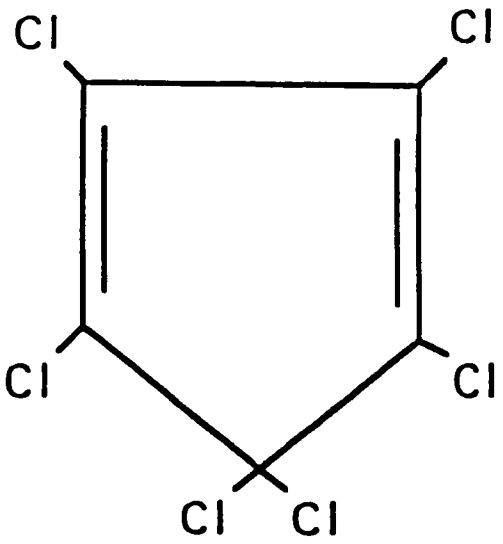
At high doses, kepone is relatively toxic to most animal species. The toxicity of kepone to humans has been investigated because of the concerns of employees and residents potentially exposed to kepone following its release into the environment at the manufacturing site mentioned above.

COMMERCIALLY IMPORTANT ROUTES OF SYNTHESIS

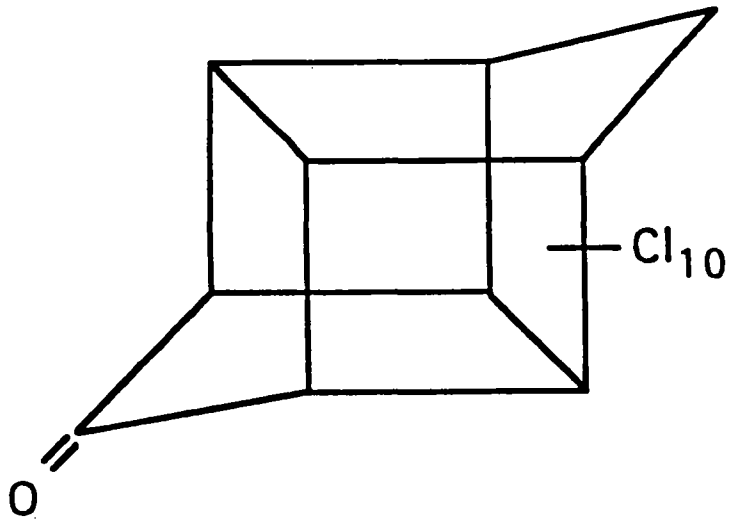
Kepone is a member of a family of chemicals created by reactions based on the chemistry of hexachlorocyclopentadiene (HCP). Known as cyclopentadienes, this group, on the whole, has general biocidal and bioactive properties. Kepone has a unique structure in that it is cubic or caged (Figure I-1). Other HCP derivatives are generally represented by planar, substituted ring variations. Their heavily chlorinated structures result in the formation of extremely stable, non-reactive molecules.

FIGURE I-1

THE CHEMICAL STRUCTURE OF
HEXACHLOROCYCLOPENTADIENE AND KEPONE



HEXACHLOROCYCLOPENTADIENE



KEPONE

Ungnade and McBee (1958) describe several reactants that can be used to synthesize kepone. These include sulfur trioxide, chlorosulfonic acid, sulfuryl chloride, or fuming sulfuric acid. When any of these are reacted with HCP at 35-80°C, the result is the formation of various sulfur-containing compounds. These compounds are then hydrolyzed, resulting in the formation of kepone hydrate in yields of 70-72%.

COMMERCIAL PRODUCTION

The first experimental synthesis of kepone was reported in the early 1950s. Commercial production first occurred in the late 1950s and continued in the United States until 1975. From the 1950s until 1975, approximately 1,600,000 kg of kepone were produced. The largest producers of kepone were Allied Chemical Corporation and, subsequently, Life Science Products Corporation, in Hopewell, Virginia. These manufacturers produced kepone from approximately 1968 until 1975. About 90% of the United States production was exported to Latin America, Africa, and Europe, and was used in the manufacture of kelevan (Epstein 1978, IARC 1979, WHO 1984).

Commercially, kepone was produced by reacting HCP with sulfur trioxide (SO_3) in the presence of a catalyst, antimony pentachloride ($SbCl_5$) (Epstein 1978, WHO 1984). The resulting products were then quenched with an aqueous alkali solution to hydrolyze the mixture to kepone. Finally, the system was neutralized with acid. As a result of the hydrolysis step, a gelatinous kepone slurry was formed. The recovery of kepone from the slurry was accomplished by centrifugation or filtration. The last step of the process was hot air drying of the recovered kepone, forming a white or tan solid (Epstein 1978).

USE AND APPLICATION

Kepone was marketed as a pesticide for the control of various insect pest species. While its use in agricultural applications was generally outside of the continental United States, kepone also found limited applications within the United States, primarily in the form of baits and wettable powders for control of various household insect pests. No information was found to document the intended mechanism of toxicity on target pest species; however, the cyclodiene groups of pesticide compounds generally act as stomach poisons.

Kepone was used in ant and cockroach traps manufactured by over 30 companies in the United States. The vast majority of these traps contained only 0.125 percent of the active ingredient and were usually designed to prevent direct human contact with the bait. Also, kepone was effective against leaf-cutting insects, but less effective against sucking insects. It was used as a fly larvicide. Other uses included control of the Colorado potato beetle, the rust mite on non-bearing citrus, and the potato wireworm and tobacco wireworm on gladioli and other plants (WHO 1984). Additionally, kepone was used in the tropics for control of

the banana root borer (NRC 1978). It was also used as a fungicide against apple scab and powdery mildew (HSDB 1992).

Following public hearings conducted to review the use and potential hazards of kepone, EPA canceled the registration for 12 products containing kepone on June 17, 1976 (IARC 1979).

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II. PHYSICAL/CHEMICAL PROPERTIES

Kepone (CAS No. 143-50-0) is a chlorinated hydrocarbon ($C_{10}Cl_{10}O$) used in the formulation of pesticide powders. It is also known by a variety of synonyms including chlordecone; decachloroketone; decachlorooctahydro-1,3,4-metheno-2H-cyclobuta[*c,d*]pentalen-2-one; decachlorotetracyclodecanone; Compound 1189; ENT 16391; GC 1189; and merex (IARC 1979). Other synonyms include 1,3,4-metheno-2H-cyclobuta(cd)pentalen-2-one,1,1a,3,3a,4,5,5,5a,5b,6-decachlorooctahydro; decachloropentacyclo[5.2.1.$0^{2,6}$ $0^{3,9}$ $0^{5,8}$]decan-4-one; decachlorotetrahydro-4,7-methanoindeneone; kepone-2-one, decachlorooctahydro; NCI-C00191; CIBA 8514; perchloropentacyclo[5.3.0.$0^{2,6}$ $0^{3,9}$ $0^{4,8}$]decan-5-one; 1,2,3,4,5,5,6,7,8,9,10,10-dodecachlorooctahydro-1,3,4-metheno-2-cyclobuta-(c,d)-pentalone;1,1a,3,3a,4,5,5,5a,5b,6-decachloro-octahydro-1,3,4-metheno-2H-cyclobuta [cd] pentalen-2-one; and 2,3,3a,4,5,6,7,7a,8,8a-decachloro-3a,4,7,7a-tetrahydro-4,7-methanoinden-1-one (HSDB 1992).

GENERAL PHYSICAL AND CHEMICAL PROPERTIES

In its manufactured form, kepone is a white to tan powder. Upon heating to 350°C, kepone sublimes with decomposition. Structurally, kepone is composed of two five-membered ring structures that are arranged in a cage-like fashion, with two chlorine atoms at each carbon position, except at the ketone position where there is a double-bond oxygen moiety attached to that carbon. It is very stable and fairly non-reactive, particularly under normal environmental conditions.

Kepone is deliquescent and forms kepone hydrate upon contact with vaporous or liquid water. The hydrated forms range from mono- to tetra-hydrates, and, ultimately, a gem-diol (in the presence of water). These characteristics have little effect on kepone's environmental persistence. Kepone has a very low vapor pressure and a relatively low solubility in water (Table II-1).

SOLUBILITY

Kepone is soluble in strongly alkaline aqueous solutions. It is readily soluble in acetone, and less soluble in benzene and light petroleum. Kepone is also soluble in alcohols, ketones, and acetic acid (IARC 1979). The reported solubility of kepone in water ranges from 0.01 mg/l (Jaber et al. 1984) to 7.6 mg/l (Howard 1991).

TABLE II-1

PHYSICAL/CHEMICAL PROPERTIES OF KEPONE

PROPERTY	DATA
Physical State (at 20°C and 760 mm Hg)	Tan to white crystalline solid.
Vapor Pressure	$< 3x10^{-7}$ mm Hg at 25°C (IARC 1979)
Vapor Density	16.4 (air=1) (Clayton and Clayton 1981)
Molecular Weight	490.6 (IARC 1979)
Melting Point	Sublimes with some decomposition at 350°C (IARC 1979).
Boiling Point	Sublimes.
Density	No reference found.
Octanol/Water Partition Coefficient	Log K_{ow} = 3.45 (Sax 1984) Log K_{ow} = 4.50 (Howard 1991)
Henry's Law Constant	2.50×10^{-8} atm-m^3/mol (Howard 1991)
Sediment/Water Partition Coefficient	$K_{sw} = 5x10^3$ (Strobel et al. 1981)
Solubility in Water	0.01 - 7.6 mg/l (Jabar et al. 1984, Howard 1991)

REACTIVITY

Kepone is not known to be reactive with acids or bases. When mixed with water, it is readily hydrated resulting in the formation of a gem-diol as shown in Figure II-1 (Guzelian 1982). Kepone solvates with water, acids, alcohols, amines, and thiols with a strained-ring carbonyl band at 5.6 μ. It absorbs light similarly to a saturated monoketone in the ultraviolet range (Ungnade and McBee 1958).

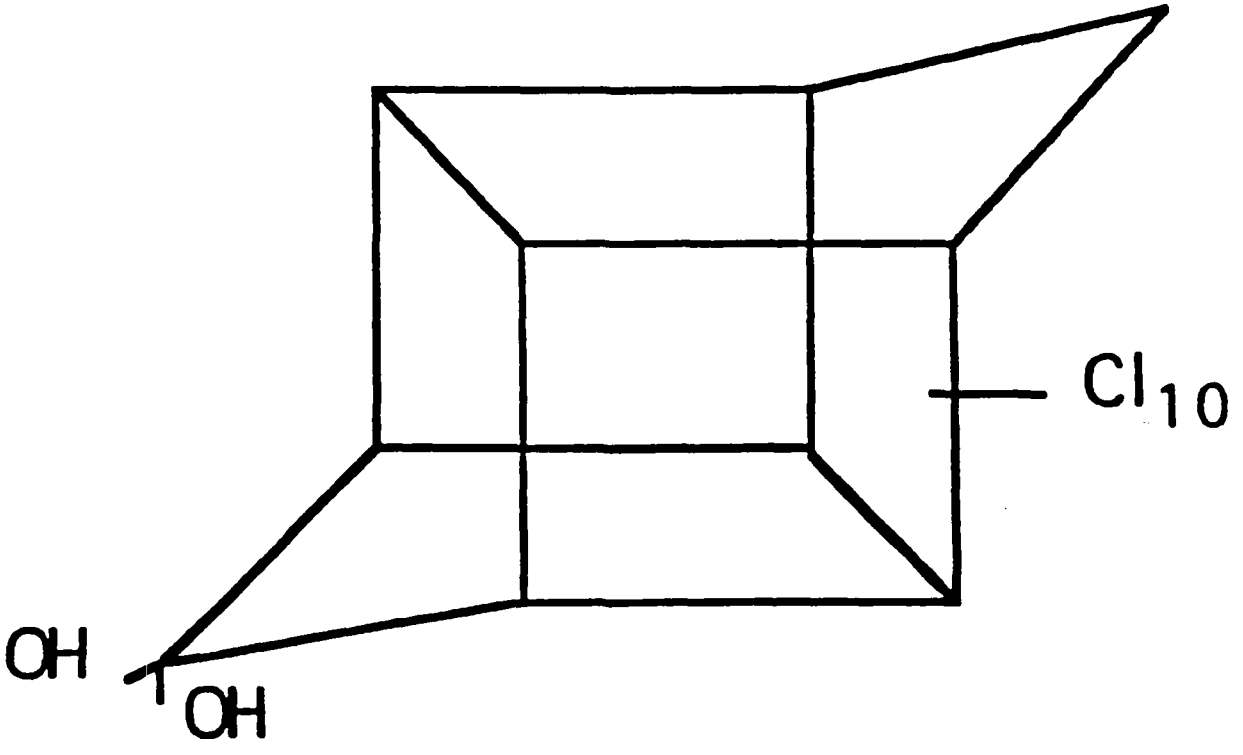
OTHER NOTABLE REACTIONS

The condensation of kepone with ethyl levulinate results in the formation of kelevan (WHO 1986).

While examining kepone in experimental aquatic systems, Huckins et al. (1982) found that methanol reacts with kepone to form a hemiketal. They also found that acetone, under reflux conditions for 18 hours, reacts with kepone to form an aldol condensation product with the empirical formula of $C_{13}H_4Cl_{10}O$. The acetone/kepone mixtures used in dosing aquatic systems were found to react, forming the same aldol condensation product after 5 weeks at room temperature. The reaction was slow to initiate (4 weeks at 20°C), but once initiated, the reaction progressed rapidly.

FIGURE II-1

THE CHEMICAL STRUCTURE OF KEPONE GEMDIOL



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III. ENVIRONMENTAL FATE AND BEHAVIOR

SOURCES OF KEPONE

Kepone is not known to occur naturally in the environment (Suta 1978). Consequently, any levels of kepone detected in the environment are presumed to have originated from the direct introduction of kepone into the environment from insecticide use, accidental or intentional release.

Insecticide Use

In the continental United States, the application and use of kepone containing products was not widespread. The indoor uses were generally limited to household ant and roach traps. Agricultural uses involved soil and spray applications. Spray applications were primarily limited to gladiolus to control corn ear worm, fall ear worm, and summer ear worm, and to non-bearing citrus trees to control rust mite (Suta 1978). Soil uses included the limited application of kepone soil baits around tobacco crops and some food crops. For those uses, kepone was generally disked into the soil or broadcast onto the soil surface prior to planting. Tobacco uses were primarily to control potato wire worm and tobacco wire worm. Use of kepone around food crops, such as endives, sweet corn, lettuce, peppers, and sugar cane, was canceled in 1968 (Suta 1978). All other remaining approved uses of kepone in the United States were discontinued in 1976 (WHO 1984).

Release from Manufacturing Sites

Kepone was evidently released into the environment from some manufacturing sites, incidental to the manufacturing process. The most studied release of kepone was from the Life Science Products Corporation manufacturing facility in Hopewell, Virginia (Suta 1978). Releases occurred over several years and resulted in the detection of kepone in the James River, its tributaries, and the lower Chesapeake Bay. Aside from this incident, there is little apparent background contamination in the continental United States. Other contamination is infrequent, is generally localized, and is usually associated with previous manufacturing sites.

Release from Degradation of Other Insecticides

Kepone has been identified as a trace contaminant in the manufacture of mirex and it is also believed to be a potential degradation product of mirex (NRC 1978). Carlson et al. (1976) studied *in situ* mirex degradation at two sites in the southeastern United States from environmental samples taken in 1974. The first site was an experimental test plot near Gulfport, Mississippi, where mirex was applied for fire ant control in 1962. The second site

was the location of a plane crash in Sebring, Florida, where mirex bait cargo was released into a pond in 1969. Carlson et al. (1976) found kepone to represent the second most abundant degradation product, with a mirex 8-monohydro derivative representing the primary product. Kepone comprised as much as 10% of the degradation products of mirex.

Kelevan was manufactured in Germany and distributed by Spiess and Sohn (Epstein 1978). Its manufacture was reported to have consumed approximately 72% of the kepone produced in the United States. Kepone is among the major degradation products of kelevan in biological and physical systems (WHO 1986). In a potato field model ecosystem, up to 7% of the environmental transformation of kelevan was to kepone (WHO 1986). No reported uses of kelevan in the continental United States were identified in preparation of this report.

FATE AND TRANSPORT OF KEPONE

Kepone is an environmentally persistent compound. Due to its relatively low aqueous solubility and very low vapor pressure, the most likely fate of kepone is ultimate storage in either soil and sediments or in biological media. Kepone is not likely to volatilize significantly from soil or water and tends to bioconcentrate in exposed animals. Kepone is readily adsorbed to soil particles and organic debris (Suta 1978). Movement of kepone in surface water systems is most likely to occur via transport of kepone adsorbed to sediments.

Air

During the period when kepone was manufactured and used in the United States, atmospheric distribution was probably a significant, but generally localized, environmental pathway. As related by Walton (1985), "[p]eople said that on a windy day when you drove by Life Science Products [Hopewell, VA] the white dust was so thick and heavy you had to cut your lights on in order to stay on the road." Today, however, ambient air is not expected to be significant in the determination of the fate and distribution of kepone. Kepone, due to its low vapor pressure ($<3x10^{-7}$mm Hg at 25°C), will not volatilize significantly to the atmosphere. Introduction into ambient air is more likely to be present in the form of contaminated dusts, which would be expected to settle out of the atmosphere relatively rapidly.

Water

Kepone's relatively low solubility in water (7 mg/l at 20-25°C) suggests that direct transport in solution (either aqueous or saline) is not a major distribution pathway. Several studies of the behavior of kepone in marine, estuarine, and aquatic systems have been conducted in Virginia in the James River/Chesapeake Bay area. These studies have focused on the

distribution and fate of kepone as a result of releases from the manufacturing operations conducted at the Life Science Products Corporation in Hopewell, Virginia.

Bender and Huggett (1984) indicated that kepone concentrations measured in James River water samples were 1 to 10 x 10^{-6} mg/l (average 6 x 10^{-6} mg/l), which was significantly lower than the reported solubility of kepone in water. The greatest concentrations were observed in areas of sediment sinks, i.e., regions of high turbidity. Thus, it appeared that the increased concentrations were associated with measurements of adsorbed kepone, not with kepone in solution. They also observed that the ultimate reduction in kepone levels in the river were generally the result of transport downstream or from burial of contaminated sediments. O'Connor et al. (1983) modeled the distribution of kepone in the James River with similar results. Kepone in the water column was predominately associated with suspended sediments, and was not due to kepone in solution. Their work highlights the importance of organic-bearing sediments in the adsorption of kepone, and the importance of these sediments in the environmental distribution of kepone.

Soil/Sediments

Soil and sediments represent a sink and are the most significant environmental media for kepone distribution. Mass transfer rates from these media to other physical media, such as water and air, are low and account for relatively little transport of the total available kepone. To the extent that soil and sediments are transported in the other media, kepone transport will occur; however, this has less to do with the characteristics of kepone than with the characteristics of the sediment-vehicle involved.

Strobel et al. (1981) evaluated the suspended sediment/water partition coefficient (K_{sw}) for kepone through field studies of the James River Estuary in Virginia. They reported a coefficient of $K_{sw}=5x10^3$, which is a reasonably accurate approximation of the observed partitioning of kepone in the field. This value is also in general agreement with the observations of Bender and Huggett (1984) and the modeling results of O'Connor et al. (1983). This value means that there is approximately a 5000 to 1 partitioning of kepone in sediments compared to kepone in solution. Strobel et al. (1981) also found that the partition coefficient remained relatively constant with sediment loading and salinity. Thus, sediments in aquatic systems represent both the major reservoir and the principal sink for kepone in the environment.

ENVIRONMENTAL DEGRADATION

In its anhydrous state, kepone is deliquescent, that is it readily absorbs moisture and forms mono- to tetra-hydrates. The keto group can then be replaced to form a gem-diol, which is relatively stable and has an enhanced solubility. This configuration imparts a weakly acidic character to kepone (Guzelian 1982). As previously noted, kepone is relatively stable in the

physical environment, and undergoes few physical/chemical transformations at ambient environmental temperatures and pressures.

Both experimental and field studies have provided evidence that kepone can slowly undergo photolytic loss of chlorine when exposed to light (Carlson et al. 1976). Although the available data are limited, photolytic decomposition may be a mechanism capable of gradually removing kepone from the environment. However, this is expected to occur only when the kepone is available to be exposed to intense sunlight over a period of years. Degradation is less likely if the kepone is adsorbed onto or covered by soil (Carlson et al. 1976).

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IV. HEALTH EFFECTS IN HUMANS AND ANIMALS

INTRODUCTION

The potential health effects associated with exposure to a particular substance are a function of both the dose to which an individual is exposed and the inherent toxicity of the material. Dose is a function of the concentration of the substance in the environmental media (i.e., exposure) and the amount of the substance that reaches the target organs of concern (i.e., toxicokinetics). This section of the report addresses the potential for human exposure to kepone, the toxicokinetics of kepone in the body, and the ability of kepone to cause organ damage. Each subsection is organized by route of exposure (inhalation, oral, dermal). Where available, human data are discussed first, followed by animal data. In the health effects subsection, data are organized by health effect and discussed in terms of acute, intermediate, and chronic exposure. The result is a comprehensive survey of the published studies related to the toxicology of kepone.

POTENTIAL FOR HUMAN EXPOSURE

The low usage of kepone in the United States has resulted in a general lack of background exposures to the United States population. Except for the James River area in Virginia, little information exists concerning kepone exposures in the population at large.

Environmental exposures to kepone are most likely to occur via ingestion or inhalation. This is primarily due to the low solubility and low vapor pressure of kepone. Exposures will most likely occur to kepone solids, to kepone adsorbed to particulate matter, or to kepone present as a contaminant of food items. Consequently, the pathways that may offer opportunities for exposures are dependent upon the environmental media.

General Population Exposures

Human breast milk was reported to have been contaminated with kepone with 3.9%, 7.5%, and 2.6% of the collected samples showing detectable levels of kepone in North Carolina, Alabama, and Georgia, respectively (Suta 1978). Kepone was not detected in milk samples from women in seven other Southeast states. Average concentrations of kepone in the positive samples were reported as < 1-3 ppb (Suta 1978). Animal studies support the neonatal transfer of kepone-contaminated milk in mice (Eroschenko and Osman 1986).

Inhalation Exposure

Inhalation exposures are likely to result in situations where kepone solids or contaminated dusts are present, but volatilizations of kepone from soil or water are not likely to contribute significantly to potential exposures. Inhalation exposures to kepone via the particulate pathway will result either in the deposition of contaminant solids directly into the respiratory system or in the subsequent ingestion of contaminant-laden mucus from the upper respiratory system. Under most inhalation exposures, the upper respiratory system will be the most likely site of initial kepone deposition.

All reported atmospheric kepone exposures have been related to workplace or manufacturing practices. As kepone has not been manufactured since 1976 in the United States, such atmospheric exposures are not expected and are included in this report only for completeness. Concentrations as high as 3 mg/m^3 were reported at the Life Science Products Corporation manufacturing plant in Hopewell, Virginia (Suta 1978). While the current National Institute for Occupational Safety and Health standard is 1 $\mu g/m^3$, neighborhood readings around the Life Science Products Corporation plant were as high as 50 $\mu g/m^3$. Readings reached as high as 30 $\mu g/m^3$ at a distance of 25 km from the plant. People near the plant may have inhaled 30-750 μg/day, whereas those living 25 km from the plant may have inhaled 0.0015-0.3 μg/day (Suta 1978).

Oral Exposure

Ingestion is the most likely route of environmental exposure to kepone. As kepone has not been manufactured in the United States since 1976, the potential for exposure is primarily limited to kepone environmental residues. Additionally, potential environmental exposures are quite limited because kepone was never widely used in the United States. However, in the vicinity of the James River, Virginia, the lower Chesapeake Bay, and proximal drainages, kepone is still present in the sediments as a result of the Life Science Products Corporation contamination. Kepone adsorbed to sediments represents the primary environmental source of kepone for introduction into biological systems in the James River/Chesapeake Bay region. Once incorporated into biological systems, kepone tends to concentrate in the food chain and ultimately magnify through predator/prey systems. No literature was identified that indicates that kepone can be incorporated into living plant tissues; however, kepone evidently can adsorb to plant surfaces.

Kepone was measured at levels ranging from 0.1 to 10 ppb in water samples taken from the James River, although no detectable levels was found in local tap water (Suta 1978). In the James River area, kepone concentrations for finfish samples averaged 0.02-2.6 ppm, oysters were found to contain 0.008-0.5 ppm, and levels in crabs were reported to be 0.10-0.34 ppm (Suta 1978). Kepone concentrations in Chesapeake Bay finfish samples averaged 0.01-0.1 ppm (Suta 1978).

Dermal Exposure

Dermal exposure to kepone is not likely to be of concern since very little kepone is absorbed via this route and because human exposure to kepone is expected to be to particulate material and not directly to kepone.

TOXICOKINETICS

The absorption, distribution, metabolism, and excretion of kepone has been examined primarily in animals, and in limited cases in humans, following inhalation, oral, and dermal exposure. The following section describes the toxicokinetics of kepone and is organized by exposure route.

Absorption

Inhalation Exposure

No studies were located regarding the absorption of kepone in humans or animals following inhalation exposure. During the manufacture of kepone at the Life Science Products Corporation manufacturing facility in Hopewell, Virginia, exposures to kepone solids, in the form of respirable dusts, contributed to the kepone body burdens in workers (Cannon et al. 1978). Human exposures to kepone were not limited to plant workers in this episode; Cannon et al. (1978) identified over 200 individuals from the Hopewell, Virginia, area with detectable blood levels of kepone ranging from 11.8 to 0.003 ppm. Documented human exposures, while highest in the Life Science Products Corporation employee group, included employee family members, cab drivers, sewage treatment plant workers, and other residents of the area (Table IV-1).

Oral Exposure

No studies were located regarding the absorption of kepone in humans following oral exposure. However, in rats dosed with 40 mg/kg kepone in corn oil by gastric intubation, kepone was found to be readily absorbed (>90%) from the gastrointestinal tract (Boylan et al. 1978, Egle et al. 1978). Huber (1965) also noted that kepone is well absorbed and distributed throughout the bodies of rats after oral administration.

TABLE IV-1

KEPONE WHOLE BLOOD LEVELS IN HUMANS[a]

GROUP	NUMBER TESTED	NUMBER WITH DETECTABLE LEVEL	PERCENTAGE WITH DETECTABLE LEVEL	RANGE OF DETECTABLE LEVEL (ppm)	MEAN OF DETECTABLE LEVEL (ppm)
Affected LSPC[b] worker	57	57	100	0.009-11.8	2.53
Unaffected LSPC worker	49	48	99	0.003-4.1	0.60
Family members, LSPC worker	32	30	94	0.003-0.39	0.10
Allied[c] Kepone worker	39	30	77	0.003-0.45	0.06
Neighborhood worker	32	23	72	0.003-0.031	0.011
Sewage treatment plant worker	10	6	60	0.004-0.014	0.008
Cab driver	5	1	20	0.003	0.003
Truck driver	2	1	50	0.004	0.004
Hopewell resident	214	40	19	0.005-0.0325	0.011

[a] Adapted from Cannon et al. (1978).
[b] Affected Life Science Products Company workers are those demonstrating clinical symptoms.
[c] Allied Chemical Corporation.

Dermal Exposure

No studies were located regarding the absorption of kepone in humans following dermal exposure. Shah et al. (1990) found that the absorption of kepone in acetone through intact, shaved, rat skin ranged from 3.9% in 6 hours to 7.9% over 72 hours. These data indicate that, under the favorable conditions of the experiment, kepone is not well-absorbed across the skin.

Distribution

Soine et al. (1982) demonstrated *in vitro* that kepone in blood will further partition by preferentially binding with high density lipoproteins (HDL) and serum albumin. This is in contrast to other common organochlorine pesticides, such as aldrin and dieldrin, which have been found by other workers (as discussed by Soine et al. 1982) to initially partition predominantly into fatty tissue and to then bind preferentially to low and very low density lipoprotein fractions in the blood. Soine et al. (1982) also demonstrated similar *in vivo* and *in vitro* distributions of kepone within the blood sera of rats and pigs (Table IV-2).

Inhalation Exposure

The distribution of kepone within the human body was reported by Cohn et al. (1978), based on samples taken from patients exposed to kepone at the Life Science Products Corporation manufacturing facility in Hopewell, Virginia. Whole blood and subcutaneous fat levels were sampled from the majority of patients (32/32 and 29/32, respectively). Liver, muscle, and gallbladder tissues were sampled from less than a third of the patients. The distribution of kepone in human tissues presented an unexpected pattern in that other organochlorine pesticide compounds are typically found to partition preferentially into adipose tissues (Table IV-3). However, Cohn et al. (1978) found that the highest levels of kepone in man occur in the liver, with the next highest levels found in subcutaneous fat. Kepone levels in muscle and gallbladder bile were similar, and whole blood had the lowest levels among the tissues and fluids examined.

Oral Exposure

Distribution of kepone following oral administration in animals is similar to Cohn et al.'s (1978) observation of kepone distribution in humans. Egle et al. (1978) observed that in four male rats orally exposed to radiolabeled kepone in a corn oil suspension via gavage, liver kepone levels were higher than fat levels, and levels in muscle, although originally much higher than gallbladder bile levels, were very similar to gallbladder bile levels by the end of the study (Table IV-4). Blood serum levels of kepone were lower than any of the tissues examined.

TABLE IV-2

PERCENTAGE OF DISTRIBUTION OF CHOLESTEROL AND [^{14}C] KEPONE IN PLASMA[a]

GROUP	TOTAL PLASMA	VLDL (d<1.006)	LDL (1.006<d<1.063)	HDL (1.063<d<1.21)	VHDL (d>1.21)
I. Rat plasma *in vivo* (n=3)					
Cholesterol	100	9	9	59	8
[^{14}C]Kepone	100	4 ± 0.6	4 ± 0.4	39 ± 2	51 ± 1
II. Rat plasma *in vitro* (n=5)					
Cholesterol	100	9	22	59	4
[^{14}C]Kepone	100	6 ± 0.3	8 ± 0.3	54 ± 1.2	31 ± 1.3
III. Human plasma *in vitro*[a]					
Cholesterol	100	7	63	20	4
[^{14}C]Kepone	100	6 ± 0.3	20 ± 1.4	30 ± 1.3	46 ± 1.7
			43 ± 1.1[b]	43 ± 1.1[b]	56 ± 0.3
IV. Pig plasma *in vitro*					
Cholesterol	100	9	53	38	0
[^{14}C]Kepone (n=2)	100	29 ± 1[c]	29 ± 1[c]	33±4[d]	33 ± 4

VLDL = very low density lipoprotein.
LDL = low density lipoprotein.
HDL = high density lipoprotein.
VHDL = very high density lipoprotein.

[a] One subject; quadruplicate incubations.
[b] d < 1.21 separated from plamsa VHDL (d > 1.21) during a single ultracentrifugation.
[c] d < 1.080.
[d] 1.080 < d < 1.21.

Adapted from Soine et al. (1982)

TABLE IV-3

PARTITIONING OF KEPONE IN HUMAN TISSUES AND FLUIDS

TISSUE/FLUID	NUMBER OF PATIENTS	RANGE (μg/g)	PARTITION	
			TISSUE/ BLOOD	RANGE
Whole Blood	32	0.6-32.0	1.0	
Liver	10	13.3-173.0	15.0	4.6-31.0
Subcutaneous fat	29	1.7-62.1	6.7	3.8-12.2
Muscle	5	1.2-11.3	2.9	1.8-4.5
Gallbladder bile	6	2.5-30.0	2.5	1.4-4.1

Source: Cohn et al. (1978)

TABLE IV-4

TIME-DEPENDENT KEPONE DISTRIBUTION IN THE SPRAGUE-DAWLEY RAT[a]

TISSUE/FLUID/ FECES	DAY 14[b]	DAY 28	DAY 49	DAY 84	DAY 182
Liver	128 ± 5.5	84.5 ± 8.3	38.8 ± 2.7	17.6 ± 0.91	3.57 ± 0.00
Fat	33.5 ± 2.6	13.3 ± 1.4	8.95 ± 1.0	1.08 ± 0.22	0.37 ± 0.02
Muscle	11.9 ± 0.8	5.23 ± 0.7	2.90 ± 0.3	0.49 ± 0.07	0.12 ± 0.00
Feces	17.1 ± 1.6	3.83 ± 1.4	1.99 ± 0.7	0.90 ± 0.19	0.21 ± 0.03
Bile	4.23 ± 0.3	2.78 ± 0.4	1.59 ± 0.7	0.70 ± 0.04	0.11 ± 0.00
Blood	2.43 ± 0.2	1.11 ± 0.1	0.71 ± 0.1	0.14 ± 0.02	0.03 ± 0.01

[a] Values based on the distribution of [^{14}C] labeled chlordecone; actual units not provided.
[b] Mean ± standard deviation

Source: Egle et al. (1978)

Dermal Exposure

No studies were located regarding kepone distribution in humans or animals following dermal exposure.

Metabolism

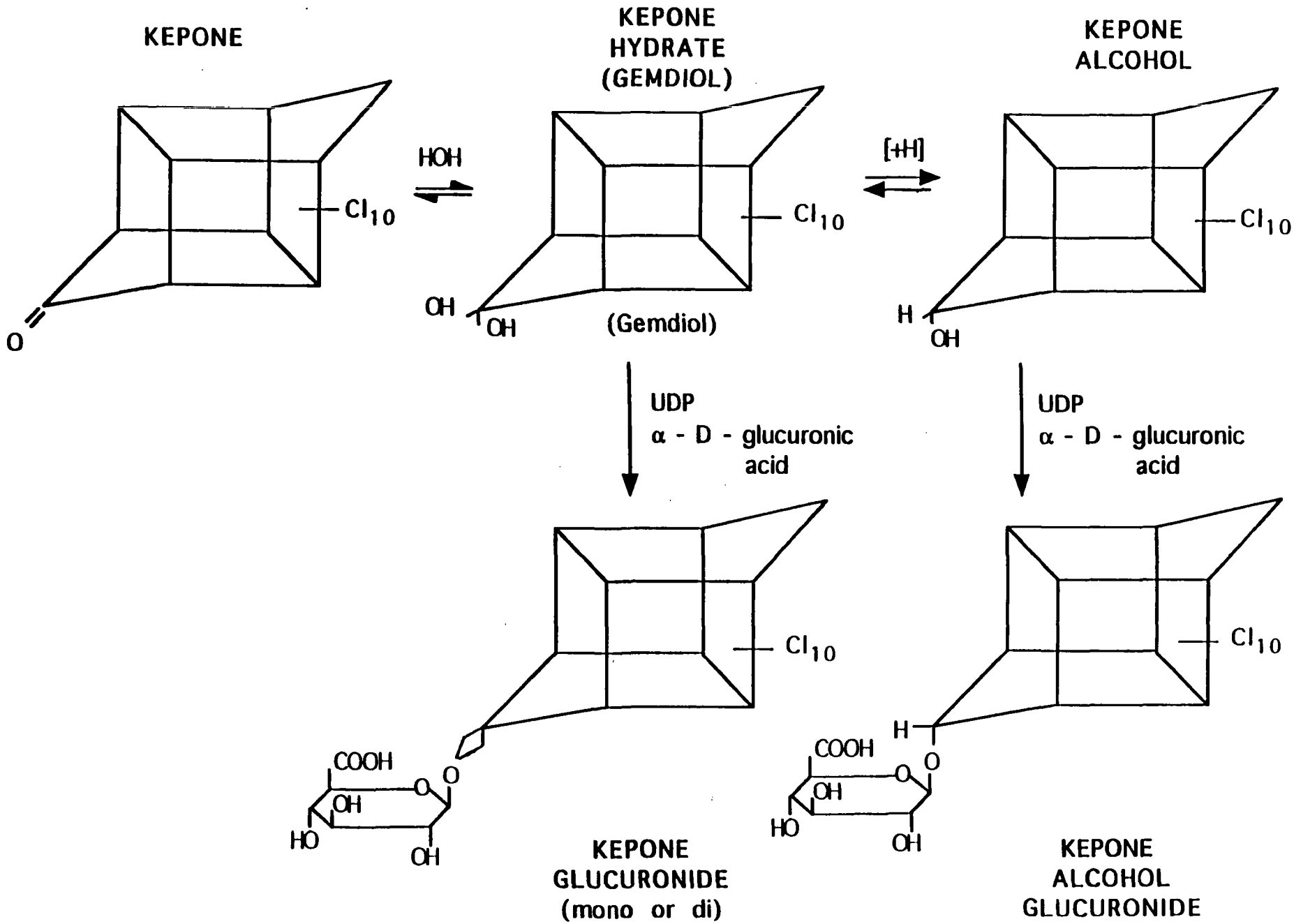
The principal metabolic products of kepone in man are kepone alcohol and kepone alcohol conjugates (Figure IV-1). These substances are produced in the liver, and, coupled with free kepone, constitute the spectrum of known metabolites found in man. In contrast, free kepone alcohol has not been detected in the rat as a product of metabolism of kepone, despite evidence that unconjugated kepone is directly eliminated in the feces via biliary and non-biliary (intestinal) pathways. Furthermore, in rats, as in man, kepone partitions, to a large extent, into the liver.

Fariss et al. (1980) conducted *in vitro* studies of human and rat bile to determine the nature and proportion of kepone metabolites produced in the liver. The human bile specimens were obtained from four patients exposed at the Life Science Products Corporation manufacturing facility in Hopewell, Virginia, and included T-tube bile obtained from the patient participating in the study by Cohn et al. (1978). Fariss et al. (1980) also collected bile from three male Sprague-Dawley rats fed a diet containing 25 ppm kepone for 2 months. Unconjugated kepone alcohol was identified in human bile, but the dominant form of kepone alcohol found in human bile was conjugated with glucuronic acid. Rat bile, in contrast to human bile, contained no unconjugated kepone alcohol and only relatively minor quantities of kepone alcohol conjugates. The ratio of kepone to kepone alcohol conjugates in human bile was found to be on the order of 1 to 2.5-3.9, and the ratio of kepone to the alcohol conjugates in rat bile was found to be 155 to 1.

The formation of kepone alcohol in rats, hamsters, guinea pigs, and gerbils was studied by Houston et al. (1981) who found that the gerbil was the only tested species in which kepone alcohol was a significant metabolite. The investigators suggested that the pathways for metabolism of kepone in gerbils more closely paralleled the human metabolic pathways because each of the other tested species apparently lacked the enzyme responsible for conversion of kepone to kepone alcohol.

Kepone metabolism in pigs was examined by Soine et al. (1983) and the reported results were similar to those obtained from human studies. In the pig, kepone is metabolized to kepone alcohol and excreted as both conjugated and unconjugated kepone alcohol. The pig partitions kepone in a manner similar to humans, e.g., in binding to serum lipoproteins and

FIGURE IV-1
KEPONE METABOLISM IN HUMANS FROM FARISS ET AL. (1980)



in distributing predominately to the liver, where the conversion to kepone alcohol occurs. Elimination of kepone in pigs is primarily accomplished via the intestinal tract and is subject to enterohepatic recycling through intestinal reabsorption (Soine et al. 1983).

Excretion

Inhalation Exposure

Excretion of kepone occurs primarily through the feces. From observations of patients exposed at the Life Science Products Corporation manufacturing facility, Cohn et al. (1978) determined that kepone concentrations in saliva, urine, and gastric juice were minimal, and levels in sweat were undetectable. Consequently, these fluids are considered to be relatively insignificant routes of elimination. Fecal elimination was concluded to be the primary route of excretion. The rate of excretion of kepone via feces is relatively slow, despite the observation that kepone levels in gallbladder bile represents a 2.5 to 1 partition over the blood compartment. Stool concentrations were only 5 percent to 10 percent of the levels found in bile (Cohn et al. 1978). The authors hypothesized that kepone excreted via gallbladder bile was reabsorbed in the intestine prior to elimination in feces. This enterohepatic recycling of kepone was demonstrated during clinical trials involving the administration of cholestyramine as an agent to bind kepone in the intestine to prohibit intestinal reabsorption (Cohn et al. 1978).

A nonbiliary pathway was also identified during these trials, as reported by Guzelian (1982a,b). In the absence of natural infusion of kepone-contaminated bile into the intestine, kepone is eliminated from the body via intestinal secretion at a rate comparable to that observed for biliary secretion of kepone and its metabolite kepone alcohol. During clinical trials, one patient agreed to the implantation of a T-tube in the common bile duct during a cholecystectomy for multiple gall stones. The tube was fitted with an inflatable distal balloon to provide an *in vivo* port for direct sampling of bile and for the introduction of bile into the intestine. The results of this experiment, shown in Table IV-5, indicated that while bile contained substantial amounts of kepone and kepone alcohol, approximately 85% of the kepone and 60% of the kepone alcohol were reabsorbed. On isolating the biliary source by not reinfusing bile, kepone levels in the stools increased and kepone alcohol levels decreased. This study demonstrated several important characteristics of kepone excretion in humans. First, in a normally functioning human system, kepone alcohol is evidently the dominant excretory product. Second, the gastrointestinal reabsorption of kepone and its alcohol is substantial. And, finally, the introduction of kepone-contaminated bile into the intestine inhibits the direct intestinal secretion of kepone. Guzelian (1982b) and Cohn et al. (1978) also found that cholestyramine, a nonabsorbable agent capable of binding kepone in the intestine, significantly accelerated the removal of kepone from the blood and increased the excretion of kepone in man and rats.

TABLE IV-5

EXCRETION OF KEPONE IN T-TUBE BILE AND FECES IN A SINGLE PATIENT

CONDITION	BILE EXCRETION (μg/24 h)		FECAL EXCRETION (μg/24 h)	
	Kepone	Kepone alcohol	Kepone	Kepone alcohol
Intact circuit (bile reinfused)	593	486	88	195
Interrupted circuit (bile diverted)	258	250	240	< 5

Source: Guzelian (1982b)

The human half-life of kepone, as presented in Table IV-6, is 165 ± 27 days (mean ± standard error of the mean) based on measurements from the blood, and 125 days (range 97 to 177 days) based on measurements of body fat, and is chiefly excreted in the feces (Cohn et al. 1978). Earlier studies by Adir et al. (1978) indicated a serum half-life of 96 ± 24 days, but this work on a cohort of Allied Chemical Company workers reported no information relating to exposure doses or durations.

Oral Exposure

No studies were located regarding the human excretion of kepone following oral exposure. Animal observations by Egle et al. (1978) identified fecal elimination as the primary route of excretion following oral exposure, and further indicated that rats did not eliminate significant amounts of kepone via respiration.

Dermal Exposure

No studies were located regarding the excretion of kepone in humans or animals following dermal exposure.

DISCUSSION OF HEALTH EFFECTS BY ROUTE OF EXPOSURE

In order to fully consider the potential for kepone to cause adverse health effects in humans, the following sections, organized according to exposure route (inhalation, oral, dermal) and then by health effect (death, systemic, immunological, neurological, developmental, reproductive, genotoxic, and carcinogenic effects), are intended to present a comprehensive survey of the published studies related to the toxicology of kepone.

Inhalation Exposure

Given the low vapor pressure of kepone, inhalation of kepone vapors is not likely to occur and would not represent a likely route of human exposure. Inhalation of kepone particulates is possible but only likely to occur at or near sites of former manufacturing facilities where kepone was produced or stored.

Death

No studies were located which permitted the evaluation of the lethality of kepone in humans and animals via inhalation exposures.

TABLE IV-6

HALF-LIFE OF KEPONE IN HUMANS

AUTHOR	TISSUE	HALF-LIFE (days)	RANGE (days)	CONFIDENCE
Cohn et al. (1978)	Blood	165 ± 27		$p < 0.005$
	Fat	125	97-177	
Guzelian (1982)	Blood	153		
	Fat	125		
Adir et al. (1978)	Blood	96 ± 24	63-148	

Systemic Effects

No studies were located which permitted the evaluation of respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, or renal effects following inhalation exposures to kepone in humans and animals.

Hepatic effects. Varying degrees of hepatomegaly have been reported in humans exposed to kepone from working at or living in the vicinity of the Life Science Products Company (LSPC). Studies to determine any effects on liver function found no significant abnormalities (Cannon et al. 1978, Taylor et al. 1978).

Dermal/Ocular. Opsoclonus or ocular flutter has been observed in humans exposed to kepone at the Life Science Products Company (Cannon et al. 1978, Taylor et al. 1978). Taylor et al. (1978) reported that these opsoclonus reports following kepone exposure were the first instance of these adverse effects occurring in humans as a consequence of known exposures to toxic materials.

Immunological Effects

No studies were located which permitted the evaluation of immunologic function following inhalation exposure to kepone in humans and animals.

Neurological Effects

Studies conducted in 1978 by Cannon et al., Taylor et al., and Cohn et al. each reported that the principal neurological effects in humans exposed to kepone at the Life Science Products Company included tremors and ataxia. These conditions coincided frequently with nervousness and exaggerated startle responses. Cannon et al. (1978) observed that these effects continued for an average of 6 weeks after exposures began, and Taylor et al. (1978) noted that these effects lasted for 5 days to 8 months after initial exposures. Routine neurologic studies have been normal or have shown only nonspecific abnormalities. Nerve biopsies indicated an increase in connective tissue but a decrease in small myelinated and unmyelinated fibers (Cannon et al. 1978, Martinez et al. 1978, Taylor et al. 1978).

Taylor (1982) reported that there had been 23 workers at the Hopewell, Virginia, chemical plant who had developed neurological manifestations after chronic exposures to kepone. The symptoms included postural and intention tremor, gait difficulty, and opsoclonus. In these workers with neurological effects, the blood serum kepone levels ranged from 2 to 33 ppm. The effects had been found to be reversible in all but one worker at the time of the Taylor report (1982).

Developmental Effects

No studies were located which permitted evaluation of fetal development following inhalation exposure to kepone in humans and animals.

Reproductive Effects

Humans exposed to kepone via inhalation have shown sperm counts which "have revealed oligospermia with abnormal and nonmotile predomination" (Cannon et al. 1978). Cohn et al. (1978) also reported decreases in sperm counts and motile sperm numbers among workers with elevated blood levels of kepone.

No studies were located which permitted the evaluation of reproductive function in animals following inhalation exposure to kepone.

Genotoxic Effects

No studies were located which permitted evaluation of mutagenicity or DNA damage following inhalation exposure to kepone in humans and animals.

Cancer

No studies were located which permitted evaluation of carcinogenicity following inhalation exposure to kepone in humans and animals.

Oral Exposure

No studies were located which permitted evaluation of the health effects of kepone in humans following oral exposure. However, much of the data obtained from individuals working at or living near the Hopewell, Virginia, manufacturing facility is relevant to this route of exposure as a result of incidental ingestion of kepone particulate matter.

Death

There are no reported incidents of human deaths occurring as a result of ingestion of kepone.

In rats the oral LD_{50} of kepone has been reported to range from 95 to 140 mg/kg body weight with some variation between the sexes (Bell 1978). When kepone was administered orally in peanut oil to male and female rats, the LD_{50} was found to be 125 mg/kg (Gaines 1969). Larson et al. (1979) conducted a series of studies to test acute, subacute, and chronic toxicities of kepone in a variety of species. Acute oral toxicities were evaluated in rats, male rabbits, and dogs, all of which received doses of kepone dissolved in corn oil and introduced via food. The oral LD_{50} for male rats was reported to be 132 mg/kg, and for females 126 mg/kg. The

LD_{50} for male rabbits was reported to be 71 mg/kg, and for dogs 250 mg/kg (Table IV-7). Egle et al. (1979) performed an acute study on Sprague-Dawley rats and reported that 60% of the animals died after single doses of 72 to 98 mg/kg, a range of doses which was $\pm$ 15% of the reported LD_{50}, approximately 85 mg/kg. While this LD_{50} is somewhat lower than that reported by Gaines (1969), the differences may be explained by the different experimental conditions, since Egle et al. (1979) dosed test animals after fasting with a corn oil/kepone solution via gastric tube.

Huber (1965) conducted a series of tests to study the effects of kepone on the laboratory mouse. At doses of 80 ppm and higher, all adults died within 32 days; at 70 ppm and higher, all juveniles died within 19 days.

The National Cancer Institute (NCI 1976) conducted a chronic effects study on rats and mice fed kepone for 80 weeks and then observed for a total of 112 and 90 weeks, respectively. The survival rates for male and female rats at high dose levels were considerably lower than for the controls. Reduced survival was noted in male mice as compared to control mice, but female mice survival was similar to that of control mice (NCI 1976).

Larson (1979) reported that rats exposed to kepone at 50 and 80 ppm in their diet died within 25 and 17 weeks, respectively.

Systemic Effects

No studies were located which permitted evaluation of systemic effects in humans following oral exposure to kepone. The available information on the health effects of ingested kepone is based on toxicological studies on experimental animals. No studies were located which permitted evaluation of respiratory, gastrointestinal, or dermal/ocular effects following oral exposure to kepone in animals.

Hematological effects. Chu et al. (1981) fed rats a diet containing 1 ppm kepone for 21 months and found no effects on hemoglobin concentration, total and differential counts of leukocytes, and bone marrow cytology. Fujimori et al. (1983) reported significant decreases in plasma glucose levels in mice exposed to 10 mg/kg/day kepone for 9 days.

Cardiovascular effects. In rats, oral intubation of 25 mg/kg kepone in corn oil for 3 days resulted in a significant decrease in calcium uptake by rat heart sarcoplasmic reticulum, suggesting that kepone may interfere with cardiac function (Kodavanti et al. 1990).

Hepatic effects. There are a number of published reports on the induction of potential adverse hepatic effects from orally administered kepone in rats, mice, and dogs. The following reports briefly highlight some of the typical adverse effects observed.

TABLE IV-7

SUMMARY OF ACUTE MAMMALIAN TOXICITY OF KEPONE

SPECIES	TEST	ROUTE/ ENTRY	DOSAGES	EFFECT	COMMENT	REFERENCE
Male Sprague-Dawley rats	Toxicity to nervous system	Ingestion	72-98 mg/kg or ±15% of LD_{50} (85 mg/kg) one time via stomach tube	Death Tremors Abnormal gait Muscle weakness observed	60% mortality between 2-5 days 95%, 3-4 h 81%, 4-5 h	Egle et al. 1979
Male Sprague-Dawley rats	Correlation between biochemical and histological indices of toxicity and tissue levels	Ingestion	1-50 mg/kg body weight	$CHCl_3$-induced liver injury	Kepone administered singly (10 mg/kg) or repetitively more effective than mirex in potentiating liver injury	Plaa et al. 1987
Rat (male)	Acute toxicity	Ingestion	132 ± 8 mg/kg	Mortality	LD_{50}, 2-7 days	Larson et al. 1979
Rat (female)	Acute toxicity	Ingestion	126 ± 6 mg/kg	Mortality	LD_{50}, 2-7 days	Larson et al. 1979
Rabbit (male)	Acute toxicity	Ingestion	71 ± 6 mg/kg	Mortality	LD_{50}, 2-11 days	Larson et al. 1979
Beagle dogs	Acute toxicity	Ingestion	250 mg/kg	Mortality	LD_{50}, 2-8 days	Larson et al. 1979
Sherman strain rats	Acute toxicity	Ingestion Dermal exposure	125 mg/kg >2000 mg/kg	Mortality	LD_{50} LD_{50}	Gaines 1969
Rabbit (male)	Acute toxicity	Percutaneous absorption	410 ± 65 mg/kg	Mortality	LD_{50}, 3-13 days	Larson et al. 1979

Several studies on kepone toxicity in rats found significant increases in liver-to-body-weight ratios in experimental animals compared to controls (Mehendale et al. 1978, Larson et al. 1979). Similar results were noted in dogs (Larson et al. 1979).

Enlarged livers were detected in rats fed kepone in the diet in several studies (Mehendale et al. 1978, Cannon and Kimbrough 1979, Mehendale 1981). Male and female rats fed a 25 ppm diet of kepone for 3 months developed enlarged livers. Four and one-half months after switching the experimental rats to the control diet, the livers in both sexes of previously exposed rats remained enlarged and showed some morphological changes (Cannon and Kimbrough 1979). Similar results were found for mice (Huber 1965, Fabacher and Hodgson 1976). Huber (1965) reported reversible liver enlargement as wells as focal necrosis, cellular hypertrophy, hyperplasia, and congestion in mice fed kepone at 30 or 100 ppm in the diet for as little as 1 week.

Hepatobiliary dysfunction was noted in rats fed a diet of 100 ppm kepone for 5 weeks; however, after the animals were restored to the control diet, recovery was observed (Mehendale 1981).

Fabacher and Hodgson (1976) reported an induction of hepatic mixed-function oxidases in male mice fed kepone in the diet at 10 and 50 ppm. Baggett et al. (1980) found increased cytochrome P-450 enzyme values and ultrastructural changes in the liver of rats at fed kepone at 200 ppm in the diet for 8 days.

Renal effects. Larson et al. (1979) detected significant organ-to-body-weight differences in the kidneys of both dogs and rats fed kepone. Beagle dogs fed kepone at 25 ppm in the diet for 127 weeks exhibited increases in kidney-to-body-weight ratios. Female rats fed kepone at 25 ppm in the diet for 24 months exhibited significant increases in kidney-to-body-weight ratios. Kidney lesions, predominantly glomerulosclerosis, were found in rats fed kepone at 25 ppm in the diet for 24 months.

Adverse kidney effects were also reported by Cannon and Kimbrough (1979). Female rats fed a diet of 25 ppm kepone for 3 months had significant increases in kidney weights and in kidney weights as a percentage of body weights when compared to controls.

Musculoskeletal effects. Muscle weaknesses developed in rats treated with kepone in corn oil at doses ranging from 72 to 98 mg/kg (Egle et al. 1979). Lack of muscle tone was detected between 6 and 21 days after dosing and became more pronounced during observation. Improvement was noted after 6 months.

Further evidence of the effects on muscle control were reported by Phillips and Eroschenko (1985), who administered kepone in 8 daily intraperitoneal injections of

17.5 mg/kg. They reported that kepone did not cause changes in the grip strength of forelimbs of mice but caused morphological changes in the ultrastructure of the skeletal muscles. These effects were generally reversed within 8 days of cessation of treatment.

Other systemic effects. Depletion of epididymal body fat stores of up to 60% was reported in male rats fed a diet containing 100 ppm kepone for 5, 15, or 20 days (Klingensmith and Mehendale 1982). This body fat depletion was suggested to result from a disruption of metabolism.

Baggett et al. (1980) found that rats fed kepone at 200 ppm in the diet for 8 days demonstrated decreased epinephrine and increased norepinephrine in adrenal glands, which was suspected to result from effects on Mg^{2+}-ATPase activity. Desaiah et al. (1977) also reported an effect of kepone on Mg^{2+}-ATPase activity, but in the livers of rats fed kepone at 50 ppm in the diet for 16 days.

Immunological Effects

No studies were located which permitted evaluation of immunological function following oral exposure to kepone in humans and animals.

Neurological Effects

No studies were located which permitted the direct evaluation of neurological function in humans following oral exposure to kepone. However, the data on neurological effects reported in individuals working in or living near the Hopewell, Virginia, manufacturing facility provide some information on the potential neurological effects associated with incidental ingestion of kepone. Data obtained for animal studies of oral exposure to kepone provide evidence of its ability to affect nerve function and impair neurobehavior.

The most common neurological effect found in animals exposed to high doses of kepone is tremor (Huber 1965, Larson et al. 1979, NCI 1976, Cannon and Kimbrough 1979, Egle et al. 1979, Baggett et al. 1980, Huang et al. 1980, Fujimori et al. 1983). Egle et al. (1979) reported tremors, abnormal gaits, and exaggerated startle responses in male Sprague-Dawley rats who received kepone in corn oil by stomach tube in doses ranging from 72 to 98 ppm. Two days after dosing, the neurological effects were most severe, followed by a period of diminishing severity. After 49 days, the neurological effects were absent (Egle et al. 1979). Baggett et al. (1980) observed hyperexcitability and persistent tremors in rats fed a diet of kepone at 200 ppm for 8 days. Huang et al. (1980) reported hyperexcitability and tremors in mice following daily oral administration of kepone at doses as low as 10 mg/kg for 9 days. Fujimori et al. (1983) reported tremors and loss of motor coordination in mice exposed to kepone at 10 mg/kg/day for 7 days. Huber (1965) reported constant tremor syndrome in mice fed a diet of kepone at 30 ppm or more prior to and for 100 days after mating; all tremors ended within 4 weeks of withdrawal of the treated diet. Cannon and Kimbrough

(1979) detected a total body tremor in rats fed kepone for 5 months; the tremors resolved when feeding was discontinued. Larson et al. (1979) noted "severe DDT-like tremors" in all species of animals chronically exposed to kepone including rats, rabbits, and dogs.

Kepone has been observed to have effects on neurobehavior in animals. Swanson and Woolley (1982) reported decreased duration of tonic full extension, total extension, and total tonus in rats following a single oral dose of kepone of 35 mg/kg body weight. Reiter and Kidd (1978) reported an exaggerated startle response and decreased ambulation in an open field in weanling rats exposed to kepone in the diet for as little as 1 week at 40 and 80 ppm, respectively. Decreased rates of response to a stimulus were observed in rats fed 1 mg/kg kepone by gastric intubation 5-6 days/week, 15 minutes prior to behavioral testing for 22 days; exposure to higher doses resulted in earlier evidence of behavioral alteration. Recovery was observed following cessation of exposure (Dietz and McMillan 1979). Squibb and Tilson (1982a) reported that exposure to kepone at 30 ppm in the diet for 60 days produced increased startle responsiveness in male rats. Increased responsiveness to an acoustic stimulus was observed following exposure to kepone at 10 ppm in the diet for 60 days.

The mechanism by which kepone is hypothesized to alter nerve cell function has been suggested to involve effects on serotonergic, GABAergic, and cholinergic neurotransmitter systems (Fujimori et al. 1982, Gerhart et al. 1985, Benet et al. 1985). Desaiah (1985) reported inhibition of neurotransmitter enzymes associated with body tremors in rats at kepone dose levels of 10, 25, and 50 mg/kg/day in a dose-dependent manner. Decreased binding of dopamine and norepinephrine to synaptosomes and reduction of uptake of dopamine and norepinephrine by synaptosomes have also been observed in kepone-exposed rats (Desaiah 1985). Kepone may also destablilize nerve cell membrane function by altering energy metabolism (Tilson and Mactutus 1982) and may block certain ion channels (Inoue et al. 1991), resulting in decreased release of neurotransmitters. Jordan et al. (1981) reported that inhibition of ATPase activity in the brain may be related to the production of tremors in rats exposed to kepone by gastric intubation.

Developmental Effects

No studies were located which permitted evaluation of fetal development following oral exposure to kepone in humans and animals. The primary developmental effect associated with maternal exposure to ketone in animals is mild impairment of behavior in the offspring of treated rats. Squibb and Tilson (1982b) reported increased negative geotaxis latencies in male offspring of rats exposed to kepone at 6 ppm in the maternal diet before and during gestation and for the first 12 days of lactation. Rosenstein et al. (1977) reported central nervous system impairment in the offspring of rats exposed to 1 or 2 mg/kg/day kepone by gastric intubation beginning on gestation day 2 and terminating at weaning. Studies in which rats were subcutaneously injected with kepone during gestation further indicate that kepone may produce long-lasting neurobehavioral changes in offspring (Tilson et al. 1982, Mactutus et al. 1982, Mactutus and Tilson 1984, 1985).

Chernoff and Rogers (1976) reported reduced fetal weight and delayed ossification in the offspring of CD rats exposed to 2 and 6 mg/kg/day kepone by gastric intubation during gestation days 7-16. More severe effects, including edema, undescended testis, enlarged renal pelvis, and enlarged cerebral ventricles were observed in offspring of rats exposed to 10 mg/kg/day kepone. However, maternal toxicity was observed at all dose levels. Increased fetal mortality and clubfoot was observed in the offspring of CD-1 mice exposed to 12 mg/kg/day kepone by gastric intubation during gestation days 7-16 (Chernoff and Rogers 1976). Squibb and Tilson (1982b) reported decreased body weight in female offspring of rats exposed to kepone at 1 ppm via the diet before and during gestation and for the first 12 days of lactation; a similar effect was observed in male offspring of rats exposed to 6 ppm.

Exposure to kepone during pregnancy appears to may affect the reproductive capability of offspring. Gellert and Wilson (1979) found that oral gavage with 15 mg/kg body weight kepone during gestation days 14 to 20 in rats produced decreased ovarian and adrenal weight, prolonged vaginal estrus, and anovulation in female offspring.

Squibb and Tilson (1982b) reported decreased body weight at 100 days, but not at 30 days, in female offspring of rats exposed to kepone at 1 ppm in the diet before and during gestation and for the first 12 days of lactation.

Reproductive Effects

No studies were located which permitted evaluation of reproductive function following oral exposure to kepone in humans and animals. Kepone administered to female rats and mice in the diet disrupts fertility. It has been suggested that kepone's effect on reproduction is due to a disturbance of central nervous system serotonergic activity or from kepone's ability to mimic estrogen and interact with the intracellular estradiol receptor (Williams and Uphouse 1991).

Swartz and Mall (1989) reported follicular toxicity (increased atresia) in CD-1 mice following oral gavage with 8 mg/kg body weight kepone for 5 consecutive days for a period of 4 weeks. Because similar effects were observed following exposure to estradiol, Swartz and Mall (1989) concluded that kepone has an estrogenic effect on the ovaries.

Good et al. (1965) reported decreases in size and number of litters in mice fed kepone added to laboratory mouse chow at 10, 17.5, 25, 30, and 37.5 ppm in the feed for 1 month prior to mating and for 5 months after mating. All dosages tested were below the lethal level. Reproduction was reduced at all dietary levels, as indicated by a decrease in size and number of litters. A series of experiments using pairs of mice (strain BALB/c) fed mixtures of kepone and laboratory mouse chow demonstrated decreased reproductive success in mice fed kepone at 5 or 10 ppm in the diet 1 month prior to mating and 4 months after mating.

Huber (1965) reported reductions in litter size, litters per pair, and young per pair in mice fed kepone at 10, 30, and 37.5 ppm for one month before mating and for 100 days after mating. A second group of mice was fed kepone at 0 and 40 ppm 2 months before mating and for 100 days after mating. Vaginal smears, hormone bioassays, histologic examinations, and mating behaviors indicated that a disturbance of the female hormonal systems had occurred. Control females had two litters in 100 days, while the females fed kepone at 40 ppm had no litters in 100 days. However, reproduction resumed within 7 weeks following the withdrawal of kepone, and the sizes of second litters following withdrawal did not differ from controls.

Cannon and Kimbrough (1979) exposed Sherman rats to kepone at 0 and 25 ppm in the diet for 3 months followed by a control diet for 4.5 months, which allowed sufficient time for two breeding cycles to be observed. Immediately after exposure was discontinued, reproduction in the kepone-fed females was completely inhibited. Nine weeks after all rats were placed on the control diet, reproductive capability in exposed female rats was partially restored. The reproductive ability of male rats fed kepone at this dose was not affected.

Linder et al. (1983) reported impaired sperm motility and viability in male rats fed kepone at 15 or 30 ppm in the diet for 90 days; the effects were reversible. Kepone exposure did not affect reproductive performance.

Injection studies in rodents also demonstrate the potential reproductive toxicity of kepone. Johnson et al. (1990) reported significantly reduced progesterone in the serum of rats undergoing normal embryo implantation 5 days after mating in female mice intraperitoneally injected with 60 mg/kg kepone. Reduced sexual activity and persistent vaginal estrus occurred in female rats exposed to 50 mg/kg kepone regardless of the stage of the estrous cycle at which the kepone was introduced (Uphouse 1985, Uphouse 1986, Brown et al. 1991). Williams and Uphouse (1991) intraperitoneally injected regularly cycling adult female rats with 25, 50, or 75 mg/kg body weight kepone on estrus, diestrus 1, or diestrus 2 and reported altered vaginal cyclicity and sexual behavior. Vaginal estrus was accelerated and the occurrence of sexual behavior was delayed, reduced, or eliminated by kepone treatment. Reductions in body weight and food intake and reduced sensitivity to progesterone could have contributed to reproductive dysfunction. Early onset of vaginal opening, persistent vaginal estrus, and anovulation have been observed in neonatal female pups injected with doses of kepone as low as 0.55 mg/kg (Gellert 1978, Sierra and Uphouse 1986).

The reproductive organs of female offspring also appear to respond to the presence of kepone in milk; accelerated sexual maturation was reported in female mice exposed to kepone through maternal milk (Eroschenko and Osman 1986).

The effect of kepone on ovulation is suspected to result from actions of kepone on estrogen target tissues within the reproductive tract (Eroschenko and Mousa 1979) or within the portions of the brain directing reproductive behavior (Reel and Lamb 1985, Brown et al. 1991). However, Cochran and Wiedow (1984) compared kepone administration by

subdermal capsule and intraperitoneal injection to estradiol and testosterone exposure in male rats and concluded that although 7.5 mg/kg kepone reduced seminal vesicle weight, it did not mimic estrogen in its effects on the reproductive system of the male rat. Kepone may also directly affect the central nervous system events that mediate female reproductive behavior through a nonsteroidal mechanism (Brown et al. 1991).

Genotoxic Effects

No studies were located which permitted evaluation mutagenicity or DNA damage in humans following oral exposure to kepone.

Simon et al. (1978) examined kepone in the dominant lethal assay in the rat and found that kepone did not induce any compound-related effects at dose of 3.6 or 11.4 mg/kg/day.

Simmons et al. (1987) tested kepone in an *in vitro* Chinese hamster ovary cytotoxicity assay and reported that kepone decreased cell density and cell viability, ATP concentration, ATP level per cell, rate of protein synthesis, and cellular protein concentration.

Cancer

No studies were located which permitted evaluation of carcinogenicity following oral exposure to kepone in humans and animals.

An NCI bioassay reported the development of cancers in laboratory test animals (Osborne-Mendel rats and hybrid mice [B6C3F1]) following the chronic administration of kepone (NCI 1976). The study involved an 80-week administration of technical grade kepone in the feed. Male rats in the low-dose group were exposed to kepone at 15 ppm for 147 days, at which time the dose was reduced to 5 ppm for the remainder of the study (413 days) due to excessive toxicity. Similar feed reductions were made for all test groups with intermediate reductions at some dose levels, and the final dose in many levels was one-sixth of the initial dose level. Except for the low-dose male rats and the high-dose male mice, all other test groups received two dose reductions at various stages of the study. Also, the high-dose male rats were only fed the kepone diet every other week during the final 75 days of the test and were fed control diet during the alternate weeks. The resulting time-weighted average doses were estimated to be 8 and 24 ppm and 18 and 26 ppm for low-dose and high-dose male and female rats, respectively, and 20 and 23 and 20 and 40 ppm for low-dose and high-dose male and female mice, respectively. The results of this study as reported by the authors (Table IV-8) indicate the formation of hepatocellular carcinomas at statistically significant levels in both male and female mice at both the estimated high- and low-dose levels. The development of hepatocellular carcinomas in male and female rats was not statistically significant in the low-dose group, but the development of hepatocellular carcinomas in female rats was reported to be significant at high-dose levels. Hepatocellular carcinoma development in the high-dose male and female rats was not significant when compared to matched controls; however, when compared to pooled controls, carcinoma development was

calculated by NCI to be significant in the high-dose males and females (Table IV-8). Due to the problems encountered with excessive exposures to kepone early in these studies, it is difficult to accurately estimate the actual doses of kepone received by any of the animals. In addition, the National Toxicology Program (NTP) has adopted a revised classification system for liver neoplasms (Maronpot et al. 1986). It is likely that the original NCI (1976) study may have significantly overestimated the cancer risk of kepone. Larson et al. (1979) found that liver samples from six rats chronically exposed to kepone in the feed (three females at 10 ppm and one female and two males at 25 ppm) revealed only equivocal evidence of carcinomatous lesions of the liver. Dogs were also chronically exposed to kepone at doses of 0, 1, 5, and 25 ppm, and histopathological examination of tissue sections of various organs, including the liver, revealed no adverse effects related to the kepone exposure (Larson et al. 1979).

Reuber (1978, 1979a,b) evaluated data on the carcinogenicity of kepone in laboratory animals, but relied on data provided by others. Both papers, as reported by the author, relied heavily on data derived from two "unpublished" studies conducted by Larson at the Medical College of Virginia (MCV). Reuber did not present necessary details concerning the protocols employed in dosing test organisms, in conducting analytical tests, in evaluating animal behaviors, or in treatment of control animals. Although there was some detail concerning the methods that were used to read tissue slides, the experiments conducted to generate those slides were not described in sufficient detail. Thus, these studies can not be scientifically evaluated. The MCV studies that formed the basis for many of Reuber's conclusions were published subsequent to Reuber's reviews, and the conclusions of the researchers involved (Larson et al. 1979) differed considerably from those reported by Reuber (1978, 1979a).

Supporting evidence of kepone's ability to promote liver tumors is found in studies conducted by Sirica et al. (1989). Subcutaneous injection of kepone in rats revealed that kepone acts largely as a liver tumor promoter rather than as a complete hepatic carcinogen in both male and female rats. Female rats appeared to be more sensitive to tumor promotion by kepone than were male rats.

Dermal Exposure

No studies were located which permitted evaluation of the health effects of kepone in humans following dermal exposure.

Death

Dermal toxicity in rabbits was determined by Larson et al. (1979) who applied kepone in corn oil to shaved skin, which was then covered. The LD_{50} for dermally dosed rabbits was determined to be 410 $\pm$65 mg/kg, and test animals at higher doses developed tremors, which were enhanced when the animals were excited (by handling). The dermal LD_{50} for kepone

NCI BIOASSAY (1976) - CARCINOGENESIS OF KEPONE

SPECIES	DOSE	EFFECTIVE NUMBER	% SURVIVAL TO STUDY TERMINATION	HEPATOCELLULAR CARCINOMAS	
				NUMBER	PERCENT
RAT (Osborne-Mendel) MALE	Pooled Control	105	63	0	0
	Matched Control	10	90	0	0
	Low	50	60	1	2
	High	44	42	3	7*
RAT (Osborne-Mendel) FEMALE	Pooled Control	100	61	0	0
	Matched Control	10	70	0	0
	Low	49	56	1	2
	High	45	40	10	22*
MOUSE (B6C3F1) MALE	Pooled Control	49	92	8	16
	Matched Control	19	90	6	32
	Low	48	58	39	81*
	High	49	50	43	88*
MOUSE (B6C3F1) FEMALE	Pooled Control	40	85	0	0
	Matched Control	10	90	0	0
	Low	50	84	26	52*
	High	49	84	23	47*

* Statistically significant, $p < 0.05$

reported by Gaines (1969) for rats was considerably higher (> 2000 mg/kg). However, kepone applied to the skin of test rats was dissolved in xylene, whereas Larson et al. (1979) employed warm corn oil as a vehicle for exposing rabbit skin.

Systemic Effects

No studies were located which permitted the evaluation of respiratory, cardiovascular, gastrointestinal, hematological, musculoskeletal, hepatic, renal, or dermal/ocular effects following dermal exposure to kepone in humans and animals.

Immunological Effects

No studies were located which permitted the evaluation of immune function following dermal exposure to kepone in humans and animals.

Neurological Effects

No studies were located which permitted the evaluation of neurological function following dermal exposure to kepone in humans and animals.

Developmental Effects

No studies were located which permitted the evaluation of fetal development following dermal exposure to kepone in humans and animals.

Reproductive Effects

No studies were located which permitted the evaluation of reproductive function following dermal exposure to kepone in humans and animals.

Genotoxic Effects

No studies were located which permitted evaluation of mutagenicity or DNA damage following oral exposure to kepone in humans and animals.

Cancer

No studies were located which permitted evaluation of carcinogenicity following inhalation exposure to kepone in humans and animals.

LEVELS IN HUMAN TISSUE AND FLUIDS ASSOCIATED WITH EFFECTS

Kepone body burdens have been reported in humans exposed to kepone from releases from the Life Science Products Corporation in Hopewell, Virginia. The reported body levels of kepone ranged from 0.003-4.1 ppm in the blood of unaffected (asymptomatic) workers to 0.009-11.8 ppm in the blood of affected (symptomatic) workers (Cannon et al. 1978). Neurological effects, including opsoclonus and tremor, hepatomegaly, and decreased sperm count and motility have been observed to be associated with the blood levels observed in affected workers (Taylor 1982, Cannon et al. 1978). However, these effects were found to be reversible, and there is no evidence that any of these symptoms reflect underlying pathological changes in the Life Science Products Corporation patient population (Guzelian 1982b).

LEVELS IN THE ENVIRONMENT ASSOCIATED WITH LEVELS IN HUMAN TISSUES AND/OR HEALTH EFFECTS

Exposures to kepone from the manufacturing facility in Hopewell, Virginia, provides an opportunity to compare environmental kepone levels with human body burdens and health effects. Kepone concentrations in the air as high as 3 mg/m^3 were reported at the plant. People near the plant may have inhaled 30-750 μg/day, whereas those living 25 km from the plant may have inhaled 0.0015-0.3 μg/day (Suta 1978). Cohn et al. (1978) sampled whole blood and subcutaneous fat from patients exposed to kepone at the facility and reported that kepone ranged from 0.6 to 32 $\mu g/g$ in whole blood and from 1.7 to 62.1 $\mu g/g$ in subcutaneous fat. The principal effects observed from these exposures were tremors and ataxia and decreased sperm count and motility (Cohn et al. 1978). As reported by Guzelian (1982 a,b), the observed effects were generally fully reversible.

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V. ENVIRONMENTAL AND REGULATORY CRITERIA

An integral part of the human health risk assessment process is the identification of levels of toxic agents that may pose potential risks to humans via specific exposure pathways. In its present form, this process involves the derivation of a reference dose (RfD), which is an estimate of the daily exposure that is unlikely to be of appreciable risk of adverse effects in a human population, and/or a cancer slope factor (CSF), which is the daily dose of a potentially carcinogenic substance that could result in one additional cancer per million exposed persons.

The EPA Risk Assessment Guidance for Superfund, Human Health Evaluation Manual (EPA 1989) recommends procedures for determining RfDs and CSFs for potentially toxic substances. The process begins with a search of the various EPA reference sources to determine whether the Agency has calculated and verified an RfD or CSF. When such criteria are not available, EPA recommends the calculation of RfDs and CSFs from data available in the open literature.

A thorough search revealed that EPA has not developed an RfD for kepone and the CSF developed for kepone appears to be unusually high. Therefore, the suitability of available toxicologic data for calculating these criteria was evaluated. Inherent in this process was the evaluation of studies in the literature for scientific method, adequacy of data, sources of interference, and suitability of application. Studies that are directly related to human toxicity were preferred over other studies. Studies utilizing common test species ranked second in utility. Studies involving uncommon test species or uncontrolled field observations were considered least valuable.

CURRENT REGULATORY STATUS

No current EPA regulatory standards concerning kepone were identified. Kepone is no longer manufactured in the U.S., and its use in the U.S. was never widespread. EPA has not developed any criteria or standards (e.g., Maximum Contaminant Levels (MCLs) or Recommended Maximum Contaminant Levels (RMCLs)) for kepone under the Safe Drinking Water Act.

The Food and Drug Administration (FDA) has established Pesticide Action Levels for kepone. These levels were originally established in response to the release of kepone into the James River, Virginia, by Life Science Products Corporation. At the time these levels were originally established, in the late 1970s, the selection of an appropriate action level was heavily influenced by the expectation of potential adverse effects at very low tolerance levels on the James River and Chesapeake Bay fishing industry. Thus, development of the tolerance levels identified by the FDA also involved consideration of economic factors on the fishing industry in addition to ecological and/or health concerns. The current action levels

for kepone, as of April 1990, are 0.4 ppm for crab meat and 0.3 ppm for fish (FDA 1990). These levels apply to the edible portions of the seafood. FDA has stated, as a matter of policy, that these action levels represent general guidance and that the FDA retains discretionary authority to determine the need for regulatory action in any particular situation. FDA has indicated that such decisions will be made on a case-by-case basis.

CARCINOGENICITY EVALUATION

EPA (1989) identified a number of reference sources that may be consulted during the process of evaluating the potential human health risks associated with exposures to toxic substances. EPA has specified in its guidance documentation that information present in the Integrated Risk Information System (IRIS) supersedes all other sources of data. To date, EPA has not published a cancer slope factor for kepone in IRIS.

Weight-of-Evidence Classification

There is no record of an EPA weight-of-evidence classification nor cancer slope factor for kepone in IRIS. However, EPA evaluated the carcinogenic potential of kepone as a hazardous substance under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA) in 1988, and at that time, classified kepone as a Group B2 carcinogen (EPA 1988). This classification was based on what was believed for be sufficient evidence of carcinogenicity from animal studies and on the absence of human carcinogenicity data. The B2 classification had not been widely disseminated within the Agency.

The International Agency for Research on Cancer (IARC 1979, 1987) classified kepone as a Group 2B compound meaning that there is no human evidence for kepone's carcinogenicity, but that there is sufficient evidence of carcinogenicity in experimental animals. The IARC Group 2B classification indicates that kepone is possibly carcinogenic to humans.

Much of the information considered by IARC was based on a review paper (Epstein 1978), much of which relied largely on unpublished studies. Part of the support for IARC's classification included Epstein's summary of a then-unpublished study of kepone conducted at the Medical College of Virginia. Epstein (1978) reported preliminary data from this study which was represented as demonstrating excess cancers in rats exposed to 25 ppm kepone in the diet. The Medical College of Virginia study was subsequently published by Larson et al. (1979) and it became apparent that evaluation of the potential carcinogenicity of kepone was not a primary goal of this study. Larson et al. (1979) briefly noted that possibly carcinomatous lesions in six of the test rats (three female rats fed 10 ppm kepone and one female and two males fed 25 ppm kepone) were independently reviewed by four pathologists who ultimately were unable to reach a consensus concerning the nature of the lesions and stated that the "possibility [of the carcinomatous nature of the liver lesions] remains an equivocal one." Therefore, the lesions should not be considered to have been cancers.

IARC (1979) also based its cancer classification, in part, on the NCI (1976) study in rats and mice. There are several concerns regarding the quality of this report which will be addressed in more detail in subsequent sections of this report.

Mutagenicity

Kepone has been tested for mutagenicity in the *Salmonella*/microsomal assay procedure (Schoeny et al. 1979). Tests were conducted involving four *Salmonella* strains, with and without metabolic activation. At all doses tested, kepone produced negative results.

Williams (1980) reviewed the results of kepone's performance in two liver culture assay systems. These included the hepatocyte primary culture/DNA repair test and the adult rat liver epithelial cell/hypoxanthine-guanine phosphoribosyl transferase mutagenesis assay. Kepone was found to be nonmutagenic in both of these *in vitro* test systems.

Simon et al. (1978) examined kepone in the dominant lethal assay in the rat and found that kepone did not induce any compound-related effects at doses of 3.6 or 11.4 mg/kg/day.

Carcinogenicity

Two published studies were identified which evaluated the carcinogenic potential of kepone following ingestion. A study by the National Cancer Institute (NCI 1976) was specifically designed to evaluate the potential carcinogenicity of kepone. A study by Larson et al. (1979) was designed to evaluate the acute, subchronic, and chronic toxicities of kepone. The data on tumor incidence from both of these studies are presented in Table V-1.

Larson et al. (1979) found that liver samples from six of the rats chronically exposed to kepone in the feed (three females at 10 ppm and one female and two males at 25 ppm) revealed only equivocal evidence of carcinomatous lesions of the liver, in spite of having been reviewed by a panel of pathologists. Both Epstein (1978) and Reuber (1978, 1979a,b) interpreted the results of Larson et al.'s study before it was published in a peer-reviewed journal and interpreted the results to be indicative that kepone is hepatocarcinogenic. However, while other papers have attributed putative carcinogenic potency to these long-term exposure studies with kepone in rats and dogs, Larson et al. (1979) were the primary researchers responsible for conducting these laboratory studies, and they clearly described the results as indicating the potential carcinogenicity of kepone as "equivocal." Therefore, the secondary interpretations of other authors (Epstein 1978, Reuber 1978, 1979a, 1979b) do not agree with the interpretations of the primary authors of these studies (Larson et al. 1979) and these data are not appropriate for the development of a cancer slope factor.

The NCI (1976) study, which reported increased incidences of liver tumors in rats and mice, utilized the then-current evaluation procedures for classification of specific hepatocellular lesions. However, the National Toxicology Program (NTP) has since developed a new system for classification of proliferative lesions of the liver, and this system has been

TABLE V-1

DEVELOPMENT OF HEPATOCELLULAR CARCINOMAS IN CHRONIC KEPONE EXPOSURE STUDIES

DOSE (ppm in feed)	LARSON ET AL. 1979 SPRAGUE-DAWLEY RATS (104 weeks)		NCI 1976 OSBORNE-MENDEL RATS (112 weeks)[a]		NCI 1976 B6C3F1 MICE (90 weeks)[a]	
	MALE	FEMALE	MALE	FEMALE	MALE	FEMALE
0	0/40	0/40	0/105 (0/10)[b]	0/100 (0/10)[b]	8/49 (6/19)[b]	0/40 (0/10)[b]
5	0/40	0/40	---	---	---	---
8	---	---	1/50	---	---	---
10	0/40	0/40	---	---	---	---
18	---	---	---	1/49	---	---
20	---	---	---	---	39/48	26/50
23	---	---	---	---	43/49	---
24	---	---	3/44	---	---	---
25	0/40	0/40	---	---	---	---
26	---	---	---	10/45	---	---
40	---	---	---	---	---	23/49
50	0/40[c]	0/40[c]	---	---	---	---
80	0/40[d]	0/40[d]	---	---	---	---

[a] Estimated time weighted average doses
[b] Pooled controls (Matched controls)
[c] All rats died within 17 weeks
[d] All rats died within 25 weeks
— Not tested

endorsed by EPA. These new guidelines distinguish between hyperplasia (a nonneoplastic response to degerative changes in the liver) and adenoma (a benign condition involving clear differentiation of cells from the surrounding tissue), which are both benign conditions as opposed to frank hepatocellular carcinomas. The NCI (1976) study included no hepatocellular adenoma category, so the reported results may have been significantly overestimated the actual cancer risk.

In addition, the NCI (1976) study contains several flaws that greatly compromise the scientific value of the results presented. There are also deficiencies in the protocol, design, and conduct of the study, which occurred in both rats and mice, which are as follows:

- Each of the groups of test animals used in the study were dosed for extended time periods at levels significantly above the Maximum Tolerated Dose (MTD).

- Test animals consistently exhibited signs of frank toxicities throughout the study. Many of the treated animals also showed loss of body weight, but these data are not reported in a table of the report so it is not possible to determine the extent of weight loss or even how frequently body weights were measured.

- Female rats and male and female mice in the high-dose groups also developed tremors during the first week of the study, and the tremors in female high-dose rats persisted until terminal sacrifice.

- Doses were administered which adversely affected survival rates of test animals due to toxicities.

- Reductions in doses were ineffective in eliminating toxic responses because of the long biological half-life of kepone in test animals and because the liver is the site of preferential concentration of kepone.

The results obtained from the use of B6C3F1 mice under the circumstances presented simply cannot be validated due to the administration of doses well in excess of the MTD and are not of sufficient quality for use in the derivation of regulatory standards. This is in spite of the extreme sensitivity of the B6C3F1 strain to liver injury from chemicals and the unusually high incidences of spontaneous neoplasms in the livers of male control mice, 16% among the pooled or room controls and 32% among the matched controls. Additionally, NCI (1976) acknowledged that "(a) the subchronic toxicity testing period was for 42 rather than 90 days; (b) the treatment period was for 18 months rather than 24 months; and (c) the number of matched controls was 10-20 rather than 50 as is currently used." NCI (1976) also acknowledged that the "changes in dosage were required to avoid toxic effects," and that "because the base design includes only two dose levels, a quantitative assessment of dose response relationships is not possible."

Sirica et al. (1989) also examined the potential carcinogenicity of kepone in a rat two-stage bioassay. In a promotion experiment, liver tumors were initiated by partial hepatectomy followed by gavage treatment with diethylnitrosamine (DEN), and then kepone was administered by biweekly subcutaneous injection. In male rats that received no DEN initiator, there was no evidence of histologically demonstrable hepatotoxic effects or hepatocarcinogenesis even after 44 weeks of kepone injections. In initiation studies, exposure to a single dose of 30 mg/kg kepone by gavage followed by treatment with sodium phenobarbital, a liver promoting agent, produced no evidence of liver tumors even after 44 weeks. These studies demonstrated that kepone is not a complete hepatic carcinogen, does not initiate liver tumors, but will promote liver tumors following chemical initiation by DEN, a potent initiator of hepatocarcinogenesis.

Dosage and Toxicity Considerations

Results derived from any testing within an animal model used to predict human responses are subject to errors associated with interspecies comparisons. Evaluations of the potential magnitude of error and the suitability of a particular animal model are based on the physiologic and metabolic differences which exist between the model used and humans. In the case of kepone, the models used to date have generally been the rat and mouse (murid rodents). For kepone, there is a fundamental metabolic difference which exists between humans and the murid model. The dominant pathway for kepone elimination in both humans and test animals is in the stool, and humans eliminate the majority of kepone in the form of kepone alcohol (Cohn et al. 1978). Murid rodents, however, lack the specific liver enzyme which forms the kepone alcohol (Guzelian 1982). Kepone intoxication produces liver effects in humans and in all other test species examined (e.g., hyperplasia). Because hepatocarcinogenicity was the tumor effect reported in the NCI (1976) bioassay, this fundamental metabolic difference causes significant concern regarding the relevance of the rat and mouse findings as applied to potential kepone carcinogenesis in humans.

Of further concern is the use of the B6C3F1 mouse strain, which has been a source of controversy for some time. This strain is particularly susceptible to the formation of spontaneous liver tumors of both benign and malignant varieties. There is considerable evidence that formation of liver cancers in this test animal may be affected by the degree of hepatic tissue damage and regeneration. This may be a particularly relevant consideration in the kepone bioassay, given the frank hepatic toxicities observed and the high levels of liver cancers noted in both the room or pooled controls and the matched controls.

Development of a Cancer Slope Factor

EPA (1988) calculated a cancer slope factor for kepone of 48 $(mg/kg/day)^{-1}$ using data from the NCI (1976) report in which male B6C3F1 mice exhibited hepatocellular carcinomas following exposure to kepone at 20 and 23 ppm in the diet for 560 days. The National Academy of Sciences (NAS 1977) used the NCI (1976) data to develop an estimated cancer

risk for kepone in drinking water. For kepone at 1 μg/liter, the risk for humans was estimated to be between 2.2 and $6x10^{-5}$, and the upper 95% confidence estimate of risk at the same concentration was reported to be 1.4 to $8x10^{-5}$. NAS (1977) also reported the upper 95% confidence estimate of lifetime cancer risk for kepone in drinking water per each μg/liter as $4.4x10^{-5}$.

For a number of reasons previously described, the NCI (1976) data do not provide rigorous support for characterizing kepone as a probable human carcinogen. Furthermore, because of the problems associated with the initial administration of excessively high doses to the test animals, the study has limited utility for characterizing dose-response relationships for kepone. In fact, the authors of the NCI study stated that "changes in dosage of chlordecone during the study were required to avoid toxic effects. For this reason, and because the basic design includes only two dose levels, a quantitative assessment of dose response relationships is not possible." As a result, a cancer slope factor for kepone should not be calculated based on the NCI (1976) study, nor on any of the studies identified in the scientific literature.

EVALUATION OF NONCARCINOGENIC EFFECTS

The EPA RfD/RfC Work Group has not developed a chronic oral reference dose (Rfd) for kepone that has been verified for inclusion into EPA's IRIS database. Therefore, an RfD for kepone was calculated in accordance with recommendations contained in the EPA's Risk Assessment Guidance for Superfund, Human Health Evaluation Manual (1989). In the absence of human data, animal studies were reviewed to identify Lowest-Observed-Adverse-Effect-Levels (LOAELs) and No-Observed-Adverse-Effect-Levels (NOAELs) following oral exposures to kepone. Insufficient human data were available from which to develop an oral reference dose; therefore, animal data were reviewed to identify the critical toxic effect of kepone used to derive the RfD. These studies are summarized in Table V-2.

Selection of Critical Data

The EPA protocol for developing an RfD requires identification of the critical toxic effect, which is the effect characterized by the lowest-observed-adverse effect level (LOAEL) and to then identify the highest dose tested that did not result in the critical adverse effect (NOAEL). This NOAEL, or in the absence of an appropriate NOAEL, the LOAEL, is then modified by application of generally order-of-magnitude uncertainty factors that reflect various types of data sets used to estimate RfDs. Figure V-1 graphically depicts the NOAELs and LOAELs identified from a survey of the experimental studies of kepone toxicity in animals.

A review of subchronic and chronic oral exposures to kepone (Table V-2) revealed that the lowest dose reported to cause an adverse effect following oral exposure to kepone is 1 ppm (0.05 mg/kg/day) administered in the maternal diet of rats during gestation and the first 12

TABLE V-2

EXPERIMENTAL STUDIES OF ORAL EXPOSURE TO KEPONE

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	EFFECT	NOAEL[a] (mg/kg/day)	LOAEL[b] (Effect)		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
1	Rat/ Sprague-Dawley	Gavage	3 days	Neurotox.	10	—	25	Jordan et al. 1981
2	Rat/ Sprague-Dawley	Oral	3 days	Neurotox.	—	—	10	Desaiah 1985
3	Rat/Long-Evans	Gavage	Postpartum days 1-4	Body Weight	—	10	—	Chernoff et al. 1979
4	Mouse/CD-1	Gavage	Postpartum days 1-4	Litter Mortality	—	—	18	Chernoff et al. 1979
5	Mouse/ICR	Oral	4 days	Neurotox.	—	—	10	Fujimori et al. 1983
6	Mouse/ICR	Gavage	4 days	Neurotox.	—	25	—	Benet et al. 1985
7	Rat/ Sprague-Dawley	Gavage	4 days	Cardiotox.	—	—	25	Kodavanti et al. 1990
8	Rat/ Sprague-Dawley	Diet	8 days	Neurotox.	—	—	0.85	Baggett et al. 1980
9	Mouse	Gavage	9 days	Neurotox.	—	10	—	Huang et al. 1980
10	Rat/ Sprague-Dawley	Diet	15 days	Hepatotox.	0.52	—	—	Curtis et al. 1979
11	Rat/ Sprague-Dawley	Diet	15 days	Hepatotox.	—	0.69	—	Curtis and Mehendale 1979

TABLE V-2

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO KEPONE
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	EFFECT	NOAEL[a] (mg/kg/day)	LOAEL[b] (Effect)		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
12	Rat/ Sprague-Dawley	Diet	15 days	Hepatotox.	0.55	---	---	Curtis and Mehendale 1980
13	Rat/ Sprague-Dawley	Diet	15 days	Impaired Metabolism	---	---	9.52	Klingensmith and Mehendale 1982
14	Rat/ Sprague-Dawley	Diet	16 days	Body Weight	---	3.6	---	Mehendale et al. 1977
15	Rat/ Sprague-Dawley	Diet	16 days	Impaired Metabolism	---	3.18	---	Desaiah et al. 1977
16	Rat	Diet	20 days	Neurotox.	---	---	9.6	USEPA unpublished 1975, as cited in Bell et al. 1979
17	Mouse/CD-1	Gavage	20 days over 4 weeks	Reprotox.	---	---	5.7	Swartz and Mall 1989
18	Rat/ Sprague-Dawley	Diet	4 weeks	Neurotox.	---	2	---	Reiter and Kidd 1978
19	Rat/ Sprague-Dawley	Diet	5 weeks	Hepatotox.	---	---	7.36	Mehendale 1981
20	Rat/Zivic-Miller	Gavage	50 days	Neurotox.	---	1	---	Dietz and McMillan 1979

TABLE V-2

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO KEPONE
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	EFFECT	NOAEL[a] (mg/kg/day)	LOAEL[b] (Effect)		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
21	Rat/Fischer 344	Diet	60 days	Neurotox.	—	0.5	—	Squibb and Tilson 1982a
22	Rat	Diet	90 days	Reprotox.	0.26	0.83	—	Linder et al. 1983
23	Rat/Sherman	Diet	3 months	Reprotox.	—	—	1.68	Cannon and Kimbrough 1979
24	Mouse	Diet	1 month before, 100 days after mating	Reprotox.	—	—	1.3	Huber 1965
25	Mouse BALB/c/ JaxGnMc	Diet	2 months before, 100 days after mating	Reprotox.	—	—	5.2	Huber 1965
26	Mouse/BALB/c	Diet	120 days	Reprotox./ Develop. tox.	—	—	0.65	Good et al. 1965
27	Rat/ Sprague-Dawley	Diet	21 months	Hepatotox.	0.07	—	—	Chu et al. 1981
28	Rat/Wistar	Diet	24 months	Reprotox./ Hepatotox.	0.275	0.55	—	Larson et al. 1979
29	Dog/Beagle	Diet	127 weeks	Renal tox.	0.125	0.625	—	Larson et al. 1979

TABLE V-2

LOWEST LEVELS OF SIGNIFICANT EXPOSURE TO KEPONE
(continued)

FIGURE KEY	SPECIES/ STRAIN	ROUTE	EXPOSURE DURATION	EFFECT	NOAEL[a] (mg/kg/day)	LOAEL[b] (Effect)		REFERENCE
						LESS SERIOUS (mg/kg/day)	SERIOUS (mg/kg/day)	
30	Rat/Fischer 344	Diet	GD[c] 1 to lactation day 12	Develop. tox. (body weight)	—	0.05[d]	—	Squibb and Tilson 1982b
31	Rat/ Sprague-Dawley	Gavage	GD[c] 2 to weaning	Develop. tox. (neurotox.)	1	—	2	Rosenstein et al. 1977
32	Rat/ Sprague-Dawley	Gavage	GD 2 to postpartum day 21	Body Weight/ Organ Weight	1.5	—	—	Chadwick et al. 1979
33	Rat/CD	Gavage	GD 7-16	Develop. tox. (fetotox.)	2	—	6	Chernoff and Rogers 1976
34	Mouse/CD-1	Gavage	GD 7-16	Develop. tox. (fetotox.)	8	—	12	Chernoff and Rogers 1976
35	Rat/ Sprague-Dawley	Gavage	GD 14-20	Develop. tox. (reprotox.)	—	—	15	Gellert and Wilson 1979

[a] A No-Observed-Adverse-Effect-Level (NOAEL) is the highest exposure level at which no harmful effects were seen in the organ system studied.

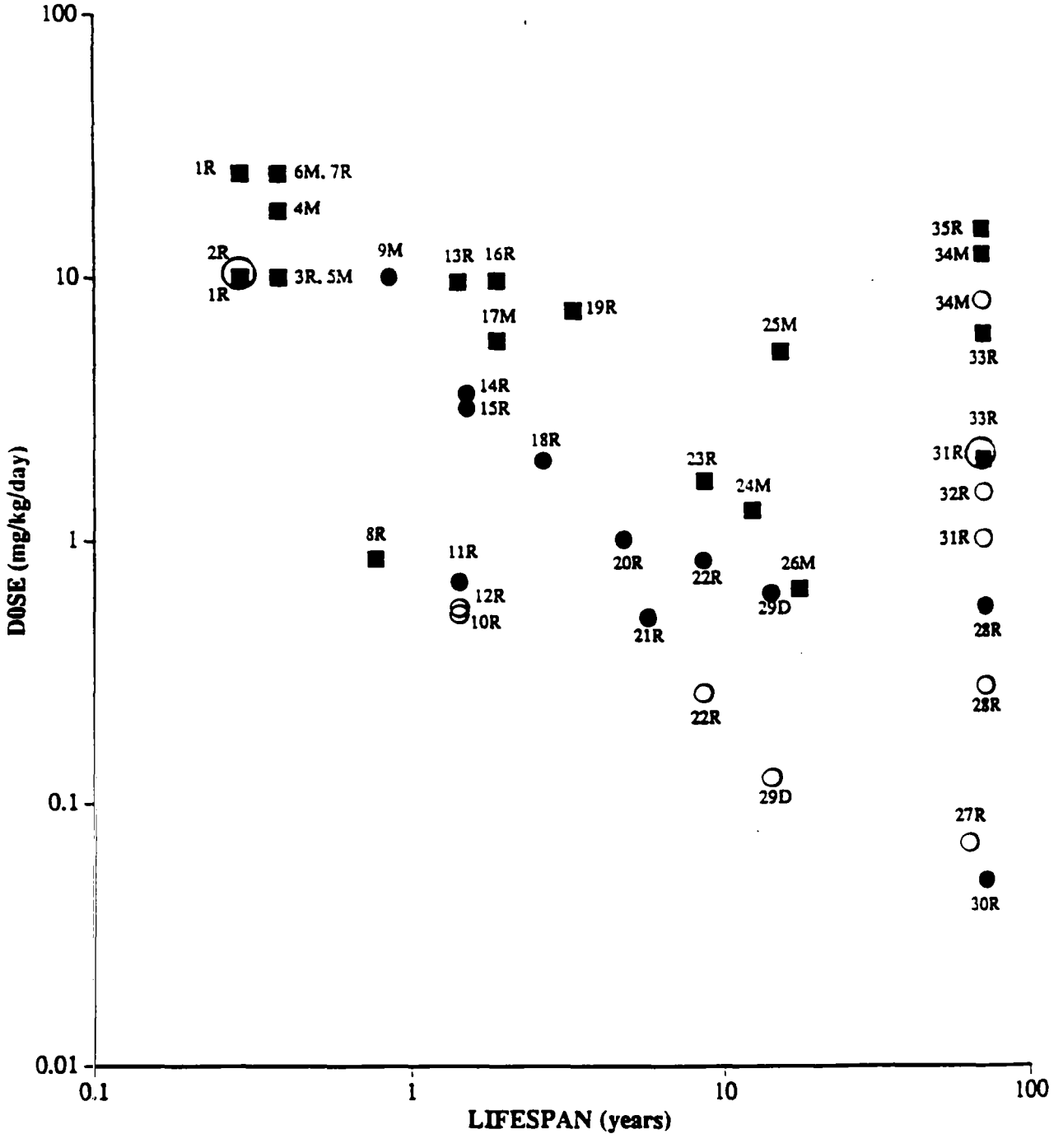
[b] A Lowest-Observed-Adverse-Effect-Level (LOAEL) is the lowest dose used in the study that caused a harmful health effect. LOAELs have been classified into "Less Serious" and "Serious" effects in order to identify the levels of exposure at which adverse health effects first appear and the reversibility or the gradation of the effects with increasing dose.

[c] GD = gestation day.

[d] Dose given to female rats only.

FIGURE V-1

EFFECT-DOSE-DURATION FOR KEPONE ANIMAL STUDIES



The data points plotted above correspond with the individual studies summarized in Table V-1. Numbers refer to figure key and R, M, and D refer to rat, mouse, and dog respectively. Effect levels are indicated by the following symbols: ■ = LOAEL (serious effects); ● = LOAEL (less serious effects); ○ = NOAEL. It should be noted that, in some cases, LOAELs for less serious effects may actually be more appropriately characterized as NOAELs and that some NOAELs may be more appropriately characterized as NOELs. Dose durations are divided by the appropriate species lifespan to yield a fraction which, when multiplied by 70 years (the assumed average human lifespan), gives the corresponding position on the X-axis.

days of lactation (Squibb and Tilson 1982b). The female offspring of Fischer 344 rats fed kepone at this dose demonstrated a 30% weight loss at 100 days of age when compared with the female offspring of control animals. Because this was the lowest dose tested, a NOAEL could not be identified. The LOAEL reported for this study is inappropriate for developing the RfD for several reasons. Inconsistent results were observed in that no effect of kepone on body weight was observed in the same female rats at 1, 7, 14, or 30 days of age, and no effect on body weight was observed at any evaluation point in male rats fed kepone at 1 ppm. Also, the average body weight of the control animals (approximately 300 g for male rats and 240 g for female rats) appeared to be high when compared with historical control data (Solleveld et al. 1984). In fact, the average body weight of the treated female rats in this study (170 g) is comparable to the body weight of 100-day old F344 female rats reported in the literature (150 g) (Solleveld et al. 1984). In a related study, Squibb and Tilson (1982a) reported that body weight loss was reversible in Fischer 344 rats 30 days after exposure to kepone at 10 ppm in the diet for 90 days. The other effect reported in the Squibb and Tilson (1982b) study is increased negative geotaxis latency in the 100-day old male offspring of rats fed kepone at 6 ppm in the diet. However, this LOAEL was considered inappropriate because a similar effect was not observed in female rats, it was the only significant effect observed in a series of neurobehavioral tests, and because the clinical significance of this effect is unclear.

Squibb and Tilson (1982a) reported the lowest dose sufficient to cause an adverse effect following oral exposure to kepone to be 10 ppm (0.5 mg/kg/day), a dose that produced a significant accentuation in the startle response of mice to an acoustic and an air puff stimulus. This LOAEL was considered inappropriate for developing the RfD because only a small number of animals was examined, the effect on startle response to an acoustic stimulus was reversible 30 days post dosing, and four other neurobehavioral tests reported in the study did not reveal an effect of kepone.

Larson et al. (1979) found the lowest dose sufficient to cause an adverse effect following oral exposure to kepone to be 10 ppm (0.55 mg/kg/day), a dose that resulted in increased liver-to-body weight ratios in female Wistar rats fed kepone for up to 2 years. A NOAEL for kepone of 5 ppm (0.275 mg/kg/day) was identified from this study which was the highest dose tested at which no significant change in liver-to-body weight ratio was observed. However, in rats returned to control diet for 4 weeks after 1 year on treated diet, the difference in means for control and 10-ppm females barely reached significance, suggesting that the observed effect was probably reversible. Larson et al. (1979) also reported a NOAEL of 5 ppm (0.125 mg/kg/day) kepone for a feeding study in beagle dogs exposed over a period of 128 weeks. Beagle dogs fed kepone at 25 ppm (0.625 mg/kg/day) in the diet for 127 weeks exhibited increases in kidney-to-body-weight ratios.

A LOAEL of 5 ppm (0.65 mg/kg/day) kepone was identified from a study by Good et al. (1965) which reported a statistically significant decrease in the size and number of litters in BALB/c mice exposed to kepone for 120 days.

Two values were considered appropriate for development of the RfD: the NOAEL of 0.125 mg/kg/day identified from the Larson et al. (1979) chronic beagle study and the LOAEL of 0.65 mg/kg/day identified from the Good et al. (1965) developmental study in mice. With the exception of increased kidney-to-body-weight ratios in rats fed kepone reported by Larson et al. (1979) and Cannon and Kimbrough (1979), no additional evidence was located to support the renal toxicity of kepone. On the other hand, several other studies reported reproductive and developmental effects in animals exposed to kepone in the diet at higher concentrations (Cannon and Kimbrough 1979, Eroschenko and Mousa 1979, Huber 1965, Rosenstein et al. 1977, Chernoff and Rogers 1976, Gellert and Wilson 1979).

Calculation of the RfD

The calculation used to develop the proposed oral RfD was carried out according to standard procedures described in EPA guidance (EPA 1986). First, doses reported as ppm in feed were converted to doses consumed in mg/kg/day. Where animal body weights and food consumption data were not reported, conversions were carried out using standard values recommended by EPA (1986). The body weight estimates for mouse and dog were 0.03 kg and 12.7 kg, respectively. Food consumption rates were estimated as a fraction of total body weight by multiplying body weight by 0.13 for mice and 0.025 for dogs.

According to EPA guidance (EPA 1986), uncertainty factors are applied to the relevant NOAEL or LOAEL to develop the RfD. An uncertainty factor of 100 was applied to the NOAEL for kepone of 0.125 mg/kg/day identified from the Larson et al. (1979) beagle study to account for interspecies extrapolation (x10) and to account for sensitive individuals in a population (x10). The resulting RfD for kepone is 0.00125 mg/kg/day.

An uncertainty factor of 1,000 was applied to the LOAEL for kepone identified from the Good et al. (1965) study, 0.65 mg/kg/day to account for interspecies extrapolation (x10), for use of a LOAEL rather than a NOAEL (x10), and for an adjustment factor to account for sensitive individuals in a population (x10). The resulting RfD for kepone is 0.00065 mg/kg/day.

Because the oral RfD derived from the Good et al. (1965) study is lower (i.e., more conservative) than the RfD derived from the Larson et al. (1979) study and reflects an adverse health effect of greater relevance to exposed individuals (effects on male reproduction have been observed in workers exposed to kepone at the Life Science Products Company), the RfD of 0.00065 mg/kg/day is recommended for kepone.

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APPENDIX G

Estimation of Environmental Concentrations in Vegetables

APPENDIX G

Estimation of Environmental Concentrations in Vegetables

Human exposure to chemicals present in soils may occur indirectly through the ingestion of vegetables grown in home gardens. The tendency for organic chemicals to bioconcentrate within produce is a function of both the physical nature of the crop and the physicochemical properties of the compounds considered.

Based on a review of the various chemicals found in soils in the vicinity of the Site, mirex and photomirex, which are highly hydrophobic compounds, are most likely to bioaccumulate in plants. Because the other chemicals evaluated do not tend to bioaccumulate into organic material to the same extent as mirex and photomirex, their contribution to overall plant uptake was assumed to be negligible. Therefore, bioaccumulation of chemicals, other than mirex and photomirex, was not evaluated.

For this assessment, chemical uptake by three classes of homegrown produce was evaluated. These three classes are:

- leafy aboveground produce (e.g., cabbage and lettuce);
- underground produce (e.g., carrots and potatoes); and
- non-leafy aboveground produce (e.g., tomatoes and cucumbers).

In determining the chemical uptake of organic chemicals by produce, three mechanisms are generally considered:

- vapor-phase uptake;
- direct deposition of particulates; and
- root uptake and stem translocation.

Because the extent of dust generation from local soils is assumed to be inconsequential, direct deposition of chemical-containing particulates onto aboveground plant surfaces is not considered. Moreover, for highly hydrophobic compounds, such as mirex and photomirex, root uptake and subsequent translocation to aboveground components of a plant are assumed to be negligible compared to other mechanisms which can impact aboveground portions of plants (USEPA 1992).

Mirex uptake by several "farm" crops (garden beans, soybeans, sorghum, and wheat) from mirex-containing soil has been documented in a greenhouse study (de la Cruz and Rajanna 1975). In this study, a field soil and loamy sand were both amended with mirex and placed in a greenhouse. Each crop investigated was grown in the amended soil for four weeks, selectively harvested (i.e., specific plant parts, such as roots, leaves and stems, were segregated for independent chemical analysis), and analyzed for mirex. While de la Cruz and Rajanna implied that the mirex detected in the different parts of these young crops (the plants were only grown for four weeks) occurred via root uptake and translocation, this uptake mechanism was not conclusively demonstrated, nor can it be deduced from the data.

It should be noted that at the time de la Cruz and Rajanna conducted their study (i.e., 1975), chemical volatilization and subsequent vapor-phase uptake was not conceptualized as a potential uptake mechanism for plants. Because the mirex was applied to a soil with low organic carbon content (0.87 percent for the field soil used, which is considerably lower than soils in a hypothetical garden with amended soil), the potential volatilization flux of mirex from the field soil would be enhanced (since there is little organic matter in the soil to which mirex can adsorb). Thus, results from this paper most likely overpredict the amount of chemical which would volatilize from soil and be found on plant surfaces. Because the study by de la Cruz and Rajanna (1975) appears to overpredict plant uptake of mirex, it provides a conservative methodology for estimating chemical accumulation in plants.

No similar experimental data are available for determining crop uptake of photomirex. Given the relative similarities in structure between mirex and photomirex, the uptake factors for photomirex are assumed to be the same as those for mirex. Based on the concentration of mirex in soil, the dry weight concentration of edible portions of plants (i.e., leaves and roots) can be estimated by the following relationship:

$$C_p = B\,C_{soil} \qquad \text{(G1)}$$

where:

C_p	=	dry weight concentration in the edible portion of plants, mg/kg
C_{soil}	=	chemical concentration in soil
B	=	soil/plant uptake factor

Using the crop concentration data from de la Cruz and Rajanna (1975), soil/plant uptake factors can be determined (Table G-1). Although de la Cruz and Rajanna did not specifically investigate home garden produce, the uptake factors derived from their study were conservatively used as a starting basis for estimating the concentration of mirex in the three classes of garden produce considered herein. From the cited study, ENVIRON utilized the mirex uptake concentrations for "field soil," realizing, however, that garden soils are often amended with various fertilizers and growth enhancers, which would result in a significantly higher organic carbon content in garden soil than that in the study's field soil.

In deriving uptake factors, ENVIRON has assumed that the root concentrations of the four crops used by de la Cruz and Rajanna are representative surrogates for underground produce. Similarly, leaf concentrations are assumed to be representative of leafy aboveground vegetables. Because de la Cruz and Rajanna did not investigate any types of non-leafy aboveground produce, ENVIRON has assumed that the uptake factor derived for leaves is applicable.

The mirex soil/plant uptake factors for the roots and leaves of the four crop types, grown at three different mirex concentrations in soil (ranging from 0.3 to 3.5 mg/kg), are presented in Table G-1. The leaf and root uptake factors for the four crop types are averaged over the three mirex soil concentrations to obtain an overall soil/plant uptake factors (B). These factors are 0.17 and 0.51 for leaves and roots, respectively.

The crops considered by de la Cruz and Rajanna (1975) are leafy aboveground produce, with the exception of garden beans. These crops are generally typified by thin outer layers. Thus, uptake by the leaves of these crops may overestimate the uptake of non-leafy produce, which tends to be much bulkier. Similarly, the root systems of the crops

TABLE G-1
Bioconcentration Factors in Leaves and Roots of Four Plants at Various Mirex Concentrations in Soil (de la Cruz and Rajanna 1975)

Plant Type	Soil Concentration (mg/kg) 3.5	0.8	0.3	Average of All Concentrations
Garden Beans				
Leaves	0.06	0.14	0.03	0.08
Roots	0.34	0.61	0.70	0.55
Soybeans				
Leaves	0.06	0.15	0.33	0.18
Roots	0.36	0.61	0.57	0.51
Sorghum				
Leaves	0.06	0.25	0.37	0.23
Roots	0.23	0.55	0.67	0.48
Wheat				
Leaves	0.05	0.23	0.30	0.19
Roots	0.33	0.34	0.77	0.48

Average Soil/Plant Uptake Factor for Leaves = 0.17
Average Soil/Plant Uptake Factor for Roots = 0.51

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investigated tend to be thin filaments. Underground produce, like its non-leafy counterparts, tends to be much bulkier. Thus, uptake by the roots of these crops may overestimate the uptake of underground produce.

USEPA (1992), in estimating the uptake of hydrophobic chemicals by both non-leafy and underground produce, applies an empirical uptake correction factor based on the geometric shape of the produce and the thickness of the skin of the plant (assumed to be 0.3 mm). This correction factor is intended to account for the physical differences between the roots and leaves of leafy crops, such as those evaluated by de la Cruz and Rajanna (1975), and bulky underground and non-leafy produce that are used to estimate exposure in this analysis. The underground and non-leafy classes of produce considered in this analysis can be approximated as spheres (potatoes and tomatoes), cones (carrots), and cylinders (cucumbers). Specifically, USEPA (1992) assumes an empirical correction factor of 0.09 for carrots, 0.03 for potatoes, and 0.01 for tomatoes and cucumbers. USEPA (1992) notes that reductions in uptake can result from the washing and peeling of produce prior to consumption. Concentration reductions from washing and peeling have not been taken into consideration in this analysis.

The garden vegetable concentrations in soils in the vicinity of the Site (i.e., on-site soils, soils adjacent to the site, and soils along the MFLBC) are estimated by combining the RME chemical concentrations in soil with the soil/plant uptake factors and empirical uptake correction factors. As noted in Equation G1, the estimated plant concentrations are on a dry weight basis, and must be converted to a wet weight basis (i.e., water mass must be considered). Based on a study by Baes et al. (1984), the wet weight fraction of underground produce, leafy aboveground produce, and non-leafy aboveground produce is 0.828, 0.936, and 0.879, respectively. In this appendix, the estimated dry and wet weight produce concentrations for a unit concentration of mirex (or photomirex) in soil (i.e., 1 mg mirex/kg soil), using the approach described above, are presented in Table G-2.

The individual vegetable concentrations used in assessing human exposure are the wet weight concentrations presented in Table G-2. Using relative human consumption rates of the three produce classes, a generic daily produce concentration can be established. Based on a study by Pao et al. (1982), the relative daily consumption of produce consists of

TABLE G-2
Dry and Wet Weight Garden Produce Concentrations
Resulting from Chemical Uptake

Chemical	Unit Soil Concentration (mg/kg)	Produce Concentration (mg/kg dry weight)[a]			Produce Concentration (mg/kg wet weight)[b]		
		Leafy Produce	Underground Produce	Non-Leafy Aboveground Produce	Leafy Produce	Underground Produce	Non-Leafy Aboveground Produce
Mirex	1.0	0.17	0.015	0.0017	0.011	0.0026	0.0002

[a] Dry weight concentrations are calculated using bioconcentration factors of 0.17 for leaves (i.e., leafy produce and non-leafy aboveground produce) and 0.51 for roots (underground produce), and uptake correction factors of 1.0 for leafy produce, 0.03 (i.e., a potato) for underground produce, and 0.01 (i.e., a tomato) for non-leafy aboveground produce.

[b] For a conversion from dry weight to wet weight concentrations, average water contents were assumed to be 0.936 for leafy produce, 0.828 for underground produce, and 0.879 for non-leafy aboveground produce.

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4.7 percent leafy produce, 9.1 percent underground produce, and 86.2 percent non-leafy aboveground produce. Applying these values to the wet weight concentrations in Table G-2, the unit concentration multiplication factor for mirex (or photomirex) in consumed produce is 0.00093 mg/kg.[1]

[1] Because chemical uptake by plants is linearly proportional to concentration in soil, the chemical concentration of mirex and photomirex in plants can be determined by multiplying the chemical concentration in soil by a unit concentration multiplication factor that is based on a mirex concentration of 1 mg/kg in soil. Based on the analysis herein, the estimated unit concentration multiplication factor for mirex in produce is 0.00093 mg/kg. This factor is equal to the sum of the following three terms: leafy produce (0.011 mg/kg)(0.047; i.e., 4.7 percent of produce intake); underground produce (0.0026 mg/kg)(0.091); and non-leafy aboveground produce (0.0002 mg/kg)(0.862).

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APPENDIX H

ODH Wildlife Sampling Results

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RICHARD F. CELESTE
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November 26, 1990

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OHIO DEPARTMENT OF HEALTH WILDLIFE SAMPLE RESULTS

Fish samples taken in 1984 and 1987 in the Middle Fork of Little Beaver Creek (MFLBC) indicated that fish in the creek contained substantial amounts of mirex from the Ruetgers-Nease Chemical Company Superfund Site in Salem, Ohio. The Ohio Department of Health issued a fish consumption advisory in 1987 and a contact advisory in 1988. This evidence raised concerns about the presence of mirex in other animals in the area of the site. In September and October 1989, the Bureau of Epidemiology and Toxicology took samples of blood and fat from raccoons and opossums at nine sites along the Middle Fork of Little Beaver Creek. These samples were taken to determine if animals other than fish had picked up mirex from their environment. The wildlife sample sites were located along the length of the creek, from near the superfund site downstream to Beaver Creek State Park.

Raccoons and opossums were chosen because these animals are the most likely to accumulate mirex in their bodies. Raccoons and opossums typically live and forage for food around creeks, rivers, lakes and wetlands. They eat a wide variety of animals and plants, including fish, crayfish, and other aquatic animals.

In areas treated with mirex to control fire ants (primarily in southern United States) residues in mammals were highest in carnivores (meat eaters, such as foxes) and insectivores (eats insects), lower in omnivores (such as raccoons and opossums) and lowest in herbivores (eats plants, such as rabbits). In other studies where mirex was applied for fire ant control, mirex concentrations were highest in omnivores and carnivores in both aquatic and terrestrial ecosystems. Most mammals living in areas treated with mirex contained mirex residues. Concentration tended to reach a maximum soon after application and declined significantly during the 12 months following.

Blood and fat samples from raccoons and opossums trapped near MFLBC contained mirex ranging from essentially none to 52.7 parts per billion (ppb) in an opossum fat sample (Table 1). Most of the samples had very low levels of mirex. The average mirex concentration in blood was 2.19 ppb and in fat, 9.17 ppb. The highest levels were in fat samples (52.7 ppb and 39.9 ppb) of animals taken closest to the site. Mirex concentrations were generally lower in animals further downstream. The variations in concentration may have been a result of animal size or age. Larger and older animals would be expected to have higher concentrations of mirex. There were also a couple of raccoon and opossum samples taken at downstream sites with slightly higher concentrations. Fish samples in this general area of the creek also contained increased levels of mirex and may have contributed to increased levels in the wildlife samples.

In published studies analyzing wildlife from areas treated with the pesticide, mirex concentrations were generally higher than what was found in samples taken along MFLBC. Mirex concentrations in some of these studies were 1000 times greater than in our samples (Table 2).

There are no federal or state regulations for allowable concentrations of mirex in sport hunted or trapped (noncommercial) wild game, however, the Federal Food and Drug Administration tolerance level for mirex in commercial meat is 100 ppb. Mirex levels in ODH's study did not approach this level. If, however, the consumer is concerned they may choose to hunt or trap another type of animal or trim the fat from these animals. Mirex concentrations would be highest in the fat. We do not believe that consumption of raccoons and opossums hunted or trapped in the MFLBC watershed poses a significant risk to human health. Mirex concentrations in raccoons and opossums in Ohio were very low compared to animals in areas of the southern U.S.

Respectfully,

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TLS:BKM/jsa/WILD.LET

TABLE 1

ODH WILDLIFE SAMPLES

Sample Site	Sample Type	Mirex Concentration (ppb)
1	A Raccoon Fat	39.9
	Serum	2.5
	B Opossum Fat	52.7
	C Opossum Fat	ND
2	A Raccoon Serum	4.5
	Fat	6.5
	B Raccoon Serum	0.7
	Fat	1.7
3	A Raccoon Serum	0.4
4	A Raccoon Serum	1.1
	Fat	ND
	B Opossum Serum	0.6
	Fat	9.6
5	A Raccoon Serum	5.9
	Fat	ND
	B Opossum Serum	6.4
	C Opossum Serum	ND
	Fat	23.7
	D Opossum Serum	8.9
6	A Opossum Serum	2.6
	Fat	ND
	B Raccoon Fat	4.3
7	A Raccoon Fat	ND
	B Opossum Fat	4.9
	C Opossum Serum	3.3
	Fat	9.5
	D Opossum Serum	7.5
	Fat	13.7
8	A Raccoon Fat	1.4
	B Raccoon Serum	5.4
	Fat	ND
	C Raccoon Fat (Road Kill)	ND
9	No Samples	
10	Raccoon Serum	0.6
	Fat	ND

Table 2
Mirex Concentration in Wildlife
from the
Southern United States

Animal	Mirex Concentration (ppm)*
Coyote fat	6.0 ppm
Opossum fat	9.5 ppm
Raccoon fat	75.9 ppm
Shrews	41.3 ppm
Frogs	9.0 ppm
Lizards	5.5 ppm
ODH-Raccoon fat	52.7 ppb**

*Parts Per Million

**Parts Per Billion

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NEWS RELEASE

RICHARD F. CELESTE
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Director of Health

November 26, 1990

FOR IMMEDIATE RELEASE

MINIMAL RISK SEEN FROM BANNED PESTICIDE FOUND IN WILDLIFE

COLUMBUS - Varying degrees of contamination from a banned pesticide found in raccoons and opossums along the Middle Fork of Little Beaver Creek in Mahoning and Columbiana counties does not pose a significant risk to human health, according to an Ohio Department of Healt study released today.

The study included nine sites along the creek watershed from the former Nease Chemical Company site near Salem to Beaver Creek State Park. Results showed varying levels of mirex in the blood and fat of the animals.

The average mirex concentration found in blood was 2.19 parts per billion while the averag level found in fat was 9.17 parts per billion. The highest levels were found in fat (52.7 ppb and 39.6 ppb) at sites closest to the former chemical company site. Concentrations tended to be much lower in downstream samples, although no pattern of decrease was noted.

While no federal standards exist for mirex levels in wildlife, the range of detectable levels of mirex found in those animals studied ranged from not being detectable to 52.7 parts per billion. These readings were considerably lower than the federal recommendation for commercial meat (100 parts per billion).

Compared to areas of the southern United States where mirex was widely used, the levels of mirex found in this wildlife study were very low.

Eating wild game is considered an individual choice and not regulated by any state or federal agency. While the Ohio Department of Health does not feel eating raccoons or opossums hunted or trapped from the creek watershed to be a significant risk, concerned consumers may want to hunt or trap animals other than raccoons or opossums or to trim fat from animals prior to eating.

Mirex has previously been found in fish samples taken from the creek and in sediment, prompting the department to issue a fish consumption advisory during 1987 and a contact advisory in 1988.

A December 1989 study of 42 people likely to be exposed to mirex showed those who had detectable levels of the pesticide in their blood either worked at the former chemical company or ate contaminated farm animal products.

A pesticide used to control imported fire ants, mirex was among pesticides produced at the site from 1961 until the Ohio Environmental Protection Agency forced the company to close 1973. The site was placed on the United States Environmental Protection Agency National Priority List (Superfund) in 1983.

Because there is no data on the effects of exposure to mirex from the environment or throu eating fish or animal products, the Ohio Department of Health recommends people to limit c reduce their exposure.

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Contact: Richard L. Wittenberg, (614) 644-8562

THE VINDICATOR - Youngstown
Friday, November 30, 1990
Pages B1 and B2

Official: Trapped animals should be safe to consume

■ Tests have found traces of mirex in animals trapped along the Middle Fork of Little Beaver Creek, but state health officials say the animals should be safe to eat.

By BOB JACKSON
VINDICATOR SALEM BUREAU

SALEM — Mirex, a suspected cancer-causing chemical, has apparently made its way into the food chain along the Middle Fork of Little Beaver Creek, an Ohio Department of Health report said.

However, the ODH says the levels of mirex found in raccoons and opossums trapped along the creek do not pose a significant health risk to humans who eat the animals.

Tracy Shelley, environmental scientist for ODH, said mirex levels in the animals ranged from non-detectable to 52.7 parts per billion. The tests checked for mirex in the blood and fat of the animals.

Averages: The average mirex concentration in blood was 2.19 ppb, and the average mirex content in fat was 9.17 ppb, Ms. Shelley said. Those numbers are well below the tolerance level of 100 ppb for commercial meats, which is what lead the ODH to issue its finding that the mirex poses no significant health risk.

"It's not necessarily a good comparison, but the commercial tolerance levels are the only thing we have to compare with. No federal standard exists," she said.

Ms. Shelley said the ODH recommends that people eat animals caught along the creek at their own risk. She said 11 raccoons and 10 opossums were taken from nine different locations along the creek. They were tested more than a year ago.

Randy Hertzer, a spokesman for the ODH, said if people are concerned, they should either hunt wild game in areas other than

Please see ANIMALS page B2

■ ANIMALS/*Safe to eat*

CONTINUED FROM PAGE B1

along the creek, or be sure to trim the fat off of any animals caught near the creek. He said mirex showed a greater tendency to settle in the fat.

Hertzer said there may be some meat contamination, but said it should be much lower than what is in the fat and blood.

Both Hertzer and Ms. Shelley said there is not enough evidence to indicate what long-term effects there are from exposure to or ingesting mirex.

Mirex is a suspected carcinogen that was manufactured at the former Nease Chemical plant on Benton Road, north of Salem. It has been detected in fish and sediment from the creek, and in some cattle that grazed near the creek.

The ODH issued a fish consumption advisory in 1987, and, in 1988, an advisory against coming into contact with the creek.

APPENDIX I

Documentation of Exposure Assumptions

Trespasser Exposure Assumptions

TABLE I-1
Intake Assumptions for Ingestion of Soil
Trespasser

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CS \times IR \times FI \times EF \times ED \times kg/10^6\ mg}{BW \times AT}$$

Parameter			Assumed Value	Reference
CS	=	Chemical Concentration in Soil (mg/kg)	See Chapter VI	
IR	=	Ingestion Rate (mg/day)	100	USEPA 1991
FI	=	Fraction Ingested from Contaminated Source (unitless)	1	USEPA 1991
EF	=	Exposure Frequency (days/yr)	24	(a)
ED	=	Exposure Duration (yrs)	9	(b)
BW	=	Body Weight (kg)	43	(c)
AT	=	Averaging Time (days) Noncarcinogens Carcinogens	 3,285 25,550	See text, Chapter VII

(a) A trespasser is assumed to trespass on to Ruetgers-Nease property approximately two times per week during summer months, or 24 days per year.
(b) Professional judgment; exposures estimated for older children and teenagers.
(c) Calculated by averaging the body weights of male and female children for the age groups 9-12 years and 12-15 years (based on data in USEPA 1990).

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TABLE I-2
Intake Assumptions for Inhalation of Air
Trespasser

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CA \times IR \times ET \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value	Reference
CA	=	Chemical Concentration in Air (mg/m^3)	See Chapter VI	
IR	=	Inhalation Rate (m^3/hr)	0.83	USEPA 1991(a)
ET	=	Exposure Time (hrs/day)	4	(b)
EF	=	Exposure Frequency (days/yr)	24	(c)
ED	=	Exposure Duration (yrs)	9	(b)
BW	=	Body Weight (kg)	43	(d)
AT	=	Averaging Time (days)		See text, Chapter VII
		Noncarcinogens	3,285	
		Carcinogens	25,550	

(a) Hourly breathing rate derived from a daily breathing rate of 20 m^3/day.
(b) Professional judgment.
(c) A trespasser is assumed to trespass on Ruetgers-Nease property approximately two times per week during summer months, or 24 days per year.
(d) Calculated by averaging the body weights of male and female children for the age groups 9-12 years and 12-15 years (based on data in USEPA 1990).

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TABLE I-3
Intake Assumptions for Incidental Ingestion of Surface Water
Trespasser

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CW \times IR \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value	Reference
CW	=	Chemical Concentration in Water (mg/L)	See Chapter VI	
IR	=	Ingestion Rate (L/event)	0.01	(a)
EF	=	Exposure Frequency (events/yr)	24	(b)
ED	=	Exposure Duration (yrs)	9	(c)
BW	=	Body Weight (kg)	43	(d)
AT	=	Averaging Time (days)		See text, Chapter VII
		Noncarcinogens	3,285	
		Carcinogens	25,550	

(a) Professional judgment. On-site drainages are small; any ingestion of on-site surface water is likely to be incidental (e.g., as a result of washing hands).

(b) A trespasser is assumed to trespass on Ruetgers-Nease property approximately two times per week during summer months, or 24 days per year.

(c) Professional judgment.

(d) Calculated by averaging the body weights of male and female children for the age groups 9-12 years and 12-15 years (based on data in USEPA 1990).

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TABLE I-4
Intake Assumptions for Ingestion of Sediment
Trespasser

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CS \times IR \times FI \times EF \times ED \times kg/10^6\ mg}{BW \times AT}$$

Parameter			Assumed Value	Reference
CS	=	Chemical Concentration in Sediment (mg/kg)	See Chapter VI	
IR	=	Ingestion Rate (mg/event)	100	USEPA 1991
FI	=	Fraction Ingested Assumed to be Contaminated Sediment (unitless)	0.25	(a)
EF	=	Exposure Frequency (events/yr)	24	(b)
ED	=	Exposure Duration (yrs)	9	(c)
BW	=	Body Weight (kg)	43	(d)
AT	=	Averaging Time (days) Noncarcinogens Carcinogens	 3,285 25,550	See text, Chapter VII

(a) On-site drainages are small. It is unlikely, therefore, that all of the soil/sediment ingested daily would come from a relatively small source. An FI of 0.25 applied to the RME soil ingestion rate is based on best professional judgment and is likely an overestimate of the extent of contact a trespasser would have with on-site sediment.

(b) A trespasser is assumed to trespass on Ruetgers-Nease property approximately two times per week during summer months, or 24 days per year.

(c) Professional judgment.

(d) Calculated by averaging the body weights of male and female children for the age groups 9-12 years and 12-15 years (based on data in USEPA 1990).

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Worker Exposure Assumptions

TABLE I-5
Intake Assumptions for Ingestion of Ground Water
Future On-site Worker

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CW \times IR \times EF \times ED}{BW \times AT}$$

		Assumed Value	Reference
CW =	Chemical Concentration in Water (mg/L)	See Chapter VI	
IR =	Ingestion Rate (L/day)	1	USEPA 1991
EF =	Exposure Frequency (days/yr)	250	USEPA 1991
ED =	Exposure Duration (yrs)	25	USEPA 1991
BW =	Body Weight (kg)	70	USEPA 1991
AT =	Averaging Time (days) Noncarcinogens Carcinogens	 9,125 25,550	See text, Chapter VII

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TABLE I-6
Intake Assumptions for Ingestion of Soil
Future On-site Worker and Worker on Adjacent Property

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CS \times IR \times 1\ kg/10^6\ mg \times FI \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value	Reference
CS	=	Chemical Concentration in Soil (mg/kg)	See Chapter VI	—
IR	=	Ingestion Rate (mg/day)	50	USEPA 1991
FI	=	Fraction Ingested from Contaminated Source (unitless)	1	--
EF	=	Exposure Frequency (days/yr)	250	USEPA 1991
ED	=	Exposure Duration (yrs)	25	USEPA 1991
BW	=	Body Weight (kg)	70	USEPA 1991
AT	=	Averaging Time (days) Noncarcinogens Carcinogens	 9,125 25,550	See text, Chapter VII

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TABLE I-7
Intake Assumptions for Inhalation of Air
Future On-site Worker

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CA \times IR \times EF \times ED}{BW \times AT}$$

Parameter		Assumed Value	Reference
CA =	Chemical Concentration in Air (mg/m^3)	See Chapter VI	
IR =	Inhalation Rate (m^3/workday)	20	USEPA 1991
EF =	Exposure Frequency (days/yr)	250	USEPA 1991
ED =	Exposure Duration (yrs)	25	USEPA 1991
BW =	Body Weight (kg)	70	USEPA 1991
AT =	Averaging Time (days)		See text, Chapter VII
	Noncarcinogens	9,125	
	Carcinogens	25,550	

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Resident Exposure Assumptions

TABLE I-8
Intake Assumptions for Ingestion of Ground Water
Future On-site Resident

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CW \times IR \times EF \times ED}{BW \times AT}$$

		Assumed Value	Reference
CW =	Chemical Concentration in Water (mg/L)	See Chapter VI	
IR =	Ingestion Rate (L/day)	2	USEPA 1991
EF =	Exposure Frequency (days/yr)	350	USEPA 1991
ED =	Exposure Duration (yrs)	30	USEPA 1991
BW =	Body Weight (kg)	70	USEPA 1991
AT =	Averaging Time (days)		See text, Chapter VII
	Noncarcinogens	10,950	
	Carcinogens	25,550	

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TABLE I-9
Intake Assumptions for Ingestion of Soil
Future On-site Resident, Resident Adjacent to Site, and Resident Along MFLBC

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CS \times IR \times 1\ kg/10^6\ mg \times FI \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value		Reference
			Adult	1-6 Years	
CS	=	Chemical Concentration in Soil (mg/kg)	See Chapter VI		—
IR	=	Ingestion Rate (mg/day)	100	200	USEPA 1991
FI	=	Fraction Ingested from Contaminated Source (unitless)	1	1	(a)
EF	=	Exposure Frequency (days/yr)	350	350	USEPA 1991
ED	=	Exposure Duration (yrs)	24	6	USEPA 1991
BW	=	Body Weight (kg)	70	15	USEPA 1991
AT	=	Averaging Time (days)			See text, Chapter VII
		Noncarcinogens	8,760	2,190	
		Carcinogens	25,550	25,550	

(a) The assumption that all soil ingested contains chemicals at the RME concentration is conservative, and particularly conservative for residents along the MFLBC, since contamination is limited to the floodplain. It is expected that most residences along the MFLBC would be located outside the floodplain, even if property extends to the creek.

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TABLE I-10
Intake Assumptions for Inhalation of Air
Future On-site Resident

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CA \times IR \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value	Reference
CA	=	Chemical Concentration in Air (mg/m^3)	See Chapter VI	
IR	=	Inhalation Rate (m^3/day)	20	USEPA 1991
EF	=	Exposure Frequency (days/yr)	350	USEPA 1991
ED	=	Exposure Duration (yrs)	30	USEPA 1991
BW	=	Body Weight (kg)	70	USEPA 1991
AT	=	Averaging Time (days)		See text, Chapter VII
		Noncarcinogens	10,950	
		Carcinogens	25,550	

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TABLE I-11
Intake Assumptions for Ingestion of Homegrown Vegetables
Future On-site Resident, Resident Adjacent to Site, and Resident Along MFLBC

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CV \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value	Reference
CV	=	Chemical Concentration in Vegetables (mg/kg)	See Chapter VI	
IR	=	Ingestion Rate (kg/day)	0.2	USEPA 1990
FI	=	Fraction Ingested from Contaminated Source (unitless)	0.40	USEPA 1990
EF	=	Exposure Frequency (days/yr)	183	USEPA 1990(a)
ED	=	Exposure Duration (yrs)	30	USEPA 1991
BW	=	Body Weight (kg)	70	USEPA 1991
AT	=	Averaging Time (days)		See text, Chapter VII
		Noncarcinogens	10,950	
		Carcinogens	25,550	

(a) In estimating the RME, USEPA 1990 recommends that the exposure duration for homegrown vegetables be 50% of the time. Expressed in days/yr, the ED = 365 days/yr x 0.5 = 183 days/yr.

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TABLE I-12
Intake Assumptions for Ingestion of Beef
Future Resident along MFLBC

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CB \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value	Reference
CB	=	Chemical Concentration in Beef (mg/kg)	See Chapter VI	
IR	=	Ingestion Rate (kg/day)	0.1	USEPA 1990
FI	=	Fraction Ingested from Contaminated Source (unitless)	0.44	USEPA 1990
EF	=	Exposure Frequency (days/yr)	350	USEPA 1991
ED	=	Exposure Duration (yrs)	30	USEPA 1991
BW	=	Body Weight (kg)	70	USEPA 1991
AT	=	Averaging Time (days) Noncarcinogens Carcinogens	 10,950 25,550	See text, Chapter VII

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TABLE I-13
Intake Assumptions for Ingestion of Milk
Future Resident along MFLBC

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CM \times IR \times FI \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value: Adult	Assumed Value: 1-6 Years	Reference
CM	=	Chemical Concentration in Milk (mg/kg)	See Chapter VI		—
IR	=	Ingestion Rate (kg/day)	0.305	0.5	(a)
FI	=	Fraction Ingested from Contaminated Source (unitless)	0.4	0.4	USEPA 1990
EF	=	Exposure Frequency (days/yr)	350	350	USEPA 1991
ED	=	Exposure Duration (yrs)	24	6	USEPA 1991
BW	=	Body Weight (kg)	70	15	USEPA 1991
AT	=	Averaging Time (days)			See text, Chapter VII
		Noncarcinogens	8,760	2,190	
		Carcinogens	25,550	25,550	

(a) Adult: USEPA 1990
Child: An age-specific ingestion rate for a child was calculated using data from Pao et al. (1982). For adults, the value of 0.305 kg/day cited by USEPA corresponds most closely to the 75th percentile whole fluid milk consumption values reported by Pao et al. (1982). As a result, the 75th percentile consumption values for 1- to 2- and 3- to 5-year old age groups in Pao et al. were averaged to derive an age-specific milk ingestion rate for a 1- to 6- year old. The resulting RME milk ingestion rate for a 1- to 6-year old was estimated to be 0.509 kg/day. Rounding to one significant figure, the IR for the child is 0.5 kg/day.

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Recreational Population Exposure Assumptions

TABLE I-14
Intake Assumptions for Ingestion of Surface Water
Recreational Visitor

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CS \times IR \times EF \times ED}{BW \times AT}$$

Parameter	Assumed Value: Current Use Upstream	Assumed Value: Future Use Upstream and Current/Future Use Downstream	References
CS = Chemical Concentration in Surface Water (mg/L)	See Chapter VI		
IR = Ingestion Rate (L/day)	0.05	0.05	(a)
EF = Exposure Frequency (days/yr)	35	70	(b)
ED = Exposure Duration (yrs)	30	30	USEPA 1991
BW = Body Weight (kg)	70	70	USEPA 1991
AT = Averaging Time (days)			See text, Chapter VII
Noncarcinogens	10,950	10, 950	
Carcinogens	25,550	25,550	

TABLE I-14
Intake Assumptions for Ingestion of Surface Water
Recreational Visitor

(a) No guidance is available on the amount of surface water that might be ingested from recreational activities such as fishing and wading. In estimating exposures while swimming, USEPA guidance (RAGS, USEPA 1989) recommends a contact rate of 50 mℓ/hour. For purposes of the assessment of the recreational visitor, an ingestion rate of 50 mℓ/day was assumed.

(b) No Advisory in Effect

The exposure frequencies (EFs) for outdoor recreational activities along the MFLBC is based on USEPA guidance and best professional judgment. The RME EF for children is based on guidance in RAGS (USEPA 1989, p. 6-43), which recommends an EF for children of 3 times per week in the fall and spring (when temperatures are above 32°F) and 5 times per week in the summer. Climate data for the Youngstown, Ohio area show that the temperature is above freezing 230 days per year, i.e., approximately 33 weeks or 8 months (U.S. Department of Commerce 1985). Using USEPA guidance, the EF for children is calculated as follows:

5 days/week x 13 weeks (summer) = 65 days
3 days/week x 20 weeks (spring and fall with temperature above 32°F) = 60 days
Total days of potential exposure = 125 days/year.

Information on the number of days spent outside by adults is unavailable, although the EF can reasonably be assumed to be smaller than for children. For the purposes of this assessment, the assumption is made that adults are potentially exposed 2 days per week for the approximately 8 months (two-thirds) of the year that the temperature is above freezing, or approximately 70 days per year.

Advisory in Effect

Because of the advisory posted along the MFLBC, the frequency of current exposure to the MFLBC upstream of Lisbon Dam is assumed to be approximately one-half that for sections of the creek downstream of the advisory and for the entire MFLBC in the future. The resulting EFs for the MFLBC within the advisory are estimated to be 60 and 35 days for the child and adult, respectively. These are reasonably conservative estimates of exposure frequency for the MFLBC based on the results of a survey conducted in September 1989 by the Ohio Department of Health (ODH 1990). The survey results showed only 12.5% of the respondents were in contact with the MFLBC once per week or more and 87.5% had contact with the MFLBC once per month or less. The ODH survey results are included in Attachment 1 to this appendix.

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TABLE I-15
Intake Assumptions for Ingestion of Sediment
Recreational Visitor

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CS \times IR \times 1\ kg/10^6\ mg \times FI \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value: Current Use Upstream		Assumed Value: Future Use Upstream and Current/Future Use Downstream		References
			Adult	1-6 Years	Adult	1-6 Years	
CS	=	Chemical Concentration in Sediment (mg/kg)	See Chapter VI				
IR	=	Ingestion Rate (mg/day)	100	200	100	200	USEPA 1991
FI	=	Fraction Ingested from Contaminated Source (unitless)	1	1	1	1	--
EF	=	Exposure Frequency (days/yr)	35	60	70	125	(a)
ED	=	Exposure Duration (yrs)	24	6	24	6	USEPA 1991
BW	=	Body Weight (kg)	70	15	70	15	USEPA 1991
AT	=	Averaging Time (days)					See text, Chapter VII
		Noncarcinogens	8,760	2,190	8,760	2,190	
		Carcinogens	25,550	25,550	25,550	25,550	

(a) See the table of Intake Assumptions for Ingestion of Surface Water -- Recreational Visitor.

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TABLE I-16
Intake Assumptions for Ingestion of Fish
Recreational Visitor

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CF \times IR \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value: Current Use Upstream	Assumed Value: Future Use Upstream and Current/Future Use Downstream	Reference
CF	=	Chemical Concentration in Fish (mg/kg)	See Chapter VI		
IR	=	Ingestion Rate (kg/meal)	0.15	0.15	(a)
EF	=	Exposure Frequency (meals/yr)	4	52	(b)
ED	=	Exposure Duration (yrs)	30	30	USEPA 1991
BW	=	Body Weight (kg)	70	70	USEPA 1991
AT	=	Averaging Time (days)			See text, Chapter VII
		Noncarcinogens	10,950	10,950	
		Carcinogens	25,550	25,550	

(a) Based on USEPA guidance that indicates that the size of fish meals ranges from 0.1 to 0.2 kg/meal, a meal size of 0.15 kg/meal is used in this assessment.

(b) The exposure frequency (EF) value (i.e., number of fish meals consumed annually) in the area of the fishing advisory (i.e., upstream of Lisbon Dam) under current conditions is based on data collected in the September 1989 ODH Survey (ODH 1990). Eighty-three percent of the respondents reported that they do not consume any fish caught in the MFLBC. The 93.5th percentile of the respondents reported eating fish caught from the MFLBC approximately 1 or 2 times in a 6-month period or less. Consequently, an exposure frequency of 1 to 2 meals/6 months or 4 meals/year approximates the 90th to 95th percentile exposure frequency and was adopted for the purposes of this assessment. The ODH survey results are included as Appendix I.

It was assumed that recreational populations would fish more often in the area downstream of the advisory. A future exposure scenario with a higher EF was also developed for the advisory area because USEPA guidance requires that hypothetical exposures be calculated as if no advisory were in place. For the purpose of this assessment, therefore, the RME EF was assumed to be 1 meal/week or 52 meals/year for ingestion of fish caught downstream of Lisbon Dam under current conditions and in the future both upstream and downstream.

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TABLE I-17
Intake Assumptions for Ingestion of Game
Recreational Visitor

Equation:

$$Intake\ (mg/kg\text{-}day) = \frac{CG \times IR \times EF \times ED}{BW \times AT}$$

Parameter			Assumed Value	Reference
CG	=	Chemical Concentration in Game (mg/kg)	See Chapter VI	
IR	=	Ingestion Rate (kg/meal)	0.15	(a)
EF	=	Exposure Frequency (meals/yr)	12	(b)
ED	=	Exposure Duration (yrs)	30	USEPA 1991
BW	=	Body Weight (kg)	70	USEPA 1991
AT	=	Averaging Time (days) Noncarcinogens Carcinogens	 10,950 25,550	See text, Chapter VII

(a) The ingestion rate for game was assumed to be the same as for fish, or 0.15 kg/meal.

(b) Exposure frequency (EF) values for current and future exposure via ingestion of game are based on data collected in the September 1989 ODH Survey (ODH 1990). Ninety-eight percent of the respondents to the ODH survey reported eating game caught in the area surrounding the MFLBC approximately once per month or less. Consequently, an EF of 1 day/month or 12 days/year approximates the 95th percentile EF and was adopted for the purposes of this assessment. The ODH survey results are included in Appendix I.

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References

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U.S. Environmental Protection Agency (USEPA). 1991. *Human health evaluation manual, supplemental guidance: Standard default exposure factors.* OSWER Directive 9285.6-03.

U.S. Environmental Protection Agency (USEPA). 1990. *Exposure factors handbook.* Exposure Assessment Group, Office of Health and Environmental Assessment, U.S. EPA, Washington, D.C. EPA/600/8-89/043.

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APPENDIX J

ODH Survey

246 N. High Street
Post Office Box 118
Columbus, Ohio 43266-0118

Telephone (614) 466-3543

RICHARD F. CELESTE
Governor

Dear Resident:

The Ohio Department of Health (ODH) is attempting to find out how many people in the Salem to Lisbon area have had exposure to the pesticide Mirex, either through the contaminated Middle Fork of the Little Beaver Creek or from the Nease Chemical Company. Mirex is classified as a potential human carcinogen. ODH and the Ohio Environmental Protection Agency are concerned about people who regularly used the upstream part of the creek because the sediments and fish are contaminated with pesticides. The Nease Chemical Company operated a plant just outside of Salem from 1961 until the EPA closed it in 1973. We are also concerned that former employees of the Nease Chemical Company may have been exposed to high levels of Mirex.

Many citizens in the area have expressed health concerns related to the pollution of the Middle Fork of the Little Beaver Creek and the Nease Chemical Company. Before we investigate any possible health problems, it is first very important to find out how many people were potentially exposed to Mirex in the creek or while working for the Nease Chemical Company. This survey is designed to assess how many people may have been exposed and for how long.

In order that the results truly represent the exposure level of the people living in the Middle Fork of the Little Beaver Creek area, it is important that each survey be completed and returned. We would like the questionnaire to be filled out by an adult member of your household, but include information about all members of the household. Please return the completed survey in the enclosed stamped return envelope as soon as possible.

Results of this survey will be released only in aggregate form. Your identity will be kept confidential. The first page of the survey containing your name and address will be separated from the remainder of the form and used only to identify who has completed the survey.

The results of this research will be made available to officials and representatives in the state and local government, members of Congress, the Ohio EPA, the U.S. EPA, the Ruetgers-Nease Company, and all interested citizens.

Your contribution to this effort is greatly appreciated. We would be happy to answer any questions you might have. Please write or call Mary Rouse in the Division of Epidemiology and Toxicology. The toll-free telephone number is 1-800-282-0546.

Sincerely,

Deborah Gray
Chief, Toxicology Branch

DLG/mar

MIDDLE FORK OF THE LITTLE BEAVER CREEK
COLUMBIANA AND MAHONING COUNTIES
OHIO DEPARTMENT OF HEALTH SURVEY

1. Please list all of the people who have lived in your household since 1961 by first and last name, sex, date of birth, and years of residence at the current address:

Name (first and last)	Sex (M/F)	Date of Birth (mo/day/yr)	Years of Residence (ex: 1961-1980)

(If you need more space, please attach another piece of paper.)

2. Please list your current address:

_____

_____

home phone number: _____

daytime phone number: _____
(if different from home)

3. Please list your former address within the Salem-Lisbon area and years of residence (if any):

Address	Years of Residence

The above information will be kept strictly confidential and separate from the rest of the survey. It is needed to identify who has completed the survey. For the remainder of the questionnaire, please identify household members by age and sex only (for example: female, 52 years old).

4. Please list present employer and all former employers since 1960 of all adult residents of this household listed in question 1, along with the dates of employment and their current age.

Employee's present age and sex Employer Dates of Employment

_____

_____

_____

_____

_____

_____

_____

5. Did you know that the Ohio Department of Health issued a fish consumption advisory for the Middle Fork of the Little Beaver Creek between Salem and Lisbon in October 1987? (circle)

1 NO
2 YES

6. From 1961 until the fish advisory was issued in October 1987, did you or anyone in your household eat fish caught from the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ---------->IF NO, SKIP FROM HERE
<-------- 2 YES TO QUESTION 10

(IF YES)

7. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

8. Using the map on page 8, put "8" by the location(s) that was most frequently fished.

9. What type of fish from this area was most often consumed?

_____

10. Since the fish advisory was issued in October 1987, have you or anyone in your household eaten fish caught from the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 14

(IF YES)

11. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

12. Using the map on page 8, put "12" by the location(s) that is most frequently fished.

13. What type of fish from this area is most often consumed?

_____

14. Since 1961, have you or any member of your household eaten game other than fish (such as deer or rabbit) hunted or trapped from the Columbiana/Mahoning County area near the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 18

(IF YES)

15. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

16. What type of game from this area is most often consumed?

_____

17. Where is the location most frequently used for hunting or trapping? If possible, use the map on page 8 and put "17" by the location(s) that is most frequently used to hunt or trap.

_____

18. Did you know that the Ohio Department of Health issued a contact advisory warning against swimming, wading, etc, for the Middle Fork of the Little Beaver Creek between Salem and Lisbon in March 1988?

1 NO
2 YES

19. From 1961 until the contact advisory was issued in March 1988, did you or members of your household swim, wade or play in the Middle Fork of the Little Beaver Creek anywhere between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 22

(IF YES)

20. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER(SPECIFY):_____

21. Using the map on page 8, put "21" by the location(s) that was most frequently used.

22. Since the contact advisory was issued in March 1988, have you or any member of your household been swimming, wading, or playing in the Middle Fork of the Little Beaver Creek anywhere between Salem and Lisbon?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 25

(IF YES)

23. Approximately how often?

1 ONCE A WEEK OR MORE
2 ONCE A MONTH
3 ONCE OR TWICE EVERY SIX MONTHS
4 ONCE A YEAR
5 LESS THAN ONCE A YEAR
6 OTHER (SPECIFY):_____

24. Using the map on page 8, put "24" by the location(s) that is most frequently used.

25. Do you live on a farm near the creek?

1 NO ----------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 31

(IF YES)

26. Do you use water from the Middle Fork of the Little Beaver Creek for irrigation?

1 NO
2 YES

27. Are any of the fields or pastures on your farm on the flood plain of MFLBC?

1 NO
2 YES

28. Are any animal or vegetable products from your farm consumed?

1 NO ---------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 31

(IF YES)

29. What type of animal and/or vegetable products from your farm are most often consumed?

_____

30. How often are any animal or vegetable products from your farm consumed?

1 ONCE OR MORE A DAY
2 THREE TO FOUR TIMES A WEEK
3 ONCE OR TWICE A WEEK
4 ONCE OR TWICE A MONTH
5 ONCE OR TWICE EVERY SIX MONTHS
6 ONCE A YEAR OR LESS
7 OTHER (SPECIFY):_____

31. Do you or anyone in your household ever eat fruit or vegetables grown in your garden or a garden in the area of the Middle Fork of the Little Beaver Creek between Salem and Lisbon?

1 NO ---------->IF NO, SKIP FROM HERE
<---------- 2 YES TO QUESTION 35

(IF YES)

32. Is water from the Middle Fork of the Little Beaver Creek used for irrigation in the garden?

1 NO
2 YES

33. What types of fruits or vegetables from that garden are most often consumed?

_____

34. How often do are any fruits or vegetables from that garden consumed?

1 ONCE A DAY OR MORE
2 THREE TO FOUR TIMES A WEEK
3 ONCE OR TWICE A WEEK
4 ONCE OR TWICE A MONTH
5 ONCE OR TWICE EVERY SIX MONTHS
6 ONCE OR LESS A YEAR
7 OTHER (SPECIFY):_____

35. What is the source of the water that comes into your home for drinking, bathing, etc?

1 CITY SUPPLY
WHAT CITY?_____
2 DUG WELL
3 DRILLED WELL
4 OTHER (SPECIFY):_____

36. Have you ever used the Middle Fork of the Little Beaver Creek or its water for anything else not already covered in this survey, such as dredging or other work-related activities?

1 NO
2 YES (SPECIFY):_____

37. Approximately how close do you live to the nearest part of the Middle Fork of the Little Beaver Creek?

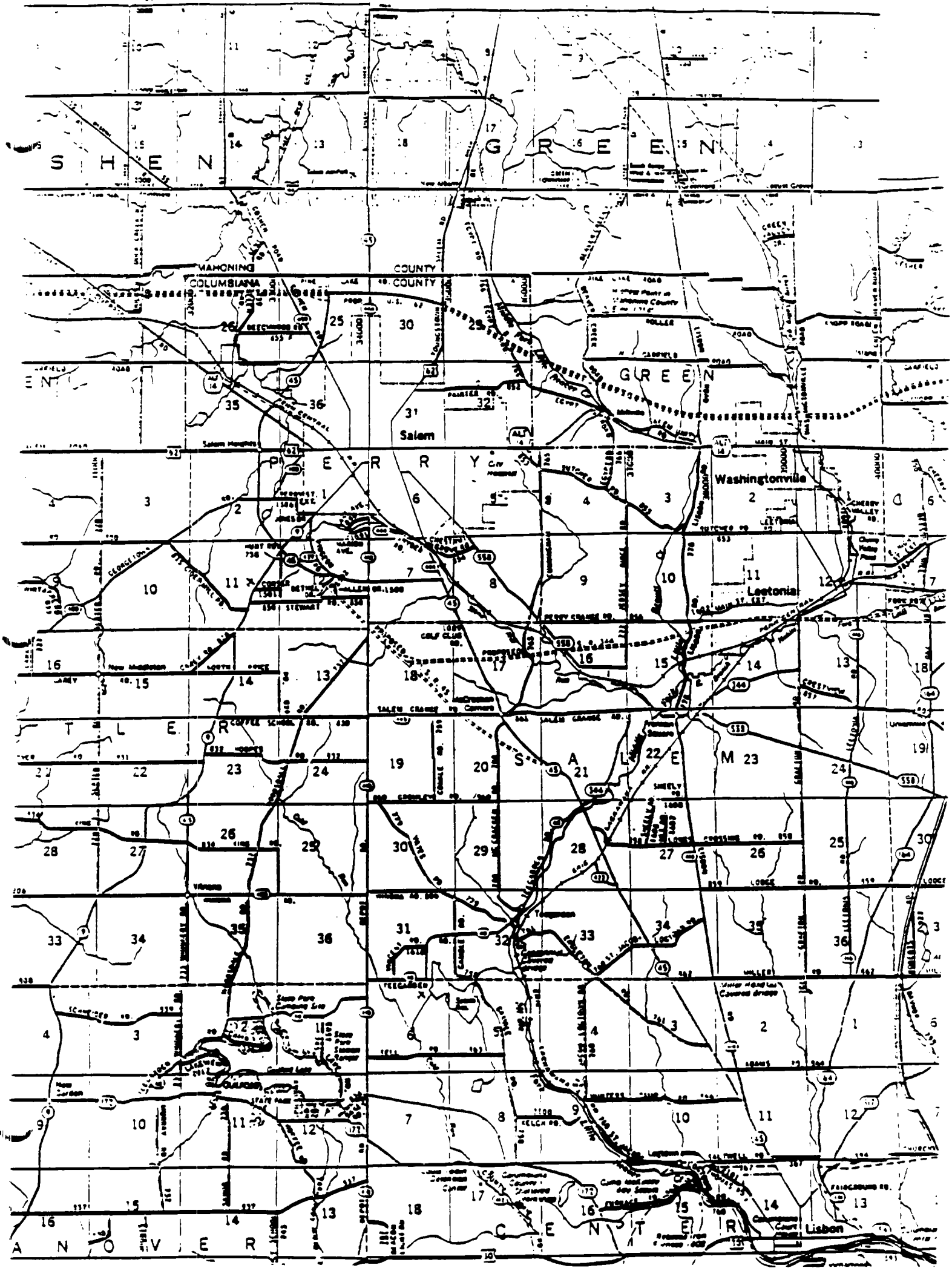
1 LIVE ON THE BANKS OR FLOODPLAIN
2 A QUARTER OF A MILE OR LESS
3 ONE QUARTER TO ONE HALF OF A MILE
4 ONE HALF TO ONE MILE
5 ONE TO TWO MILES
6 MORE THAN TWO MILES

38. Using the map on page 8, put an "X" at the location where you liv

Is there anything else you would like to tell us regarding the Middl Fork of the Little Beaver Creek, the Nease Chemical Company, or an possible health conditions you feel may be related to the above? If so please use this space and the back of this sheet as needed.

Also, any comments you wish to make that you think may help us i future efforts to better understand the situation will be appreciated either here or in a separate letter.

MAR/mar
8/89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ANOVER

INTEROFFICE COMMUNICATION

To: Rob Winters, Environ Corporation
From: Mary Rouse Martin, Ohio Department of Health
Date: February 13, 1991
Subject: Ruetgers-Nease Superfund Site Survey Data

Rob,

Please find attached a copy of the results of the survey conducted by the Ohio Department of Health, Bureau of Epidemiology and Toxicology in September 1989 regarding potential exposure to contaminants from the Ruetgers-Nease Superfund Site. This information was reported in condensed form in "Assessment of Exposure to Mirex Associated with the Nease Chemical Company Superfund Site in Salem, Columbiana County, Ohio" from the Ohio Department of Health, October 4, 1990. The tables summarize the information regarding the five potential pathways for exposure under study by the Ohio Department of Health. Please note that the results for the fish consumption and sediment contact are for the period of time since the advisories were issued in 1987 and 1988, respectively.

If can be of further assistance, please give me a call.

Sincerely,
Mary Rouse Martin

Mary Rouse Martin
Epidemiologist
Bureau of Epidemiology and Toxicology
Ohio Department of Health
P.O. Box 118
Columbus, Ohio 43266-0118
614/644-6447

APPENDIX H: FREQUENCY OF ACTIVITIES RELATED TO THE NEASE SUPERFUND SITE AND MFLBC AS REPORTED AMONG 200 SURVEY RESPONDENTS

Table I: Frequency of Contact with MFLBC and its Sediments Among 200 Survey Respondents

contact frequency	number of respondents	percentage
none	119	59.5
<1/year	16	8.0
1/year	7	3.5
1-2/6 months	21	10.5
1/month	12	6.0
≥1/week	25	12.5

Table II: Frequency of Consuming Game Hunted or Trapped from MFLBC Area Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	135	67.5%
>1/year	12	6.0%
1/year	17	8.5%
1-2/6 months	22	11.0%
1/month	10	5.0%
≥1/week	4	2.0%

Table III: Frequency of Consuming Fish from MFLBC Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	166	83.0%
>1/year	6	3.0%
1/year	4	2.0%
1-2/6 months	11	5.5%
1/month	6	3.0%
≥1/week	7	3.5%

Table IV: Frequency of Consumption of Garden Products Irrigated by MFLBC Water for Crop Irrigation Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	183	91.5%
1/year	1	0.5%
1-2/6 months	2	1.0%
1-2/month	2	1.0%
1-2/week	3	1.5%
3-4/week	9	4.5%

From: Rouse Martin M, Shelley TL, Mortensen BK. Assessment of Exposure to Mirex Associated with the Nease Chemical Company Superfund Site in Salem, Columbiana County, Ohio. Ohio Department of Health. October 4, 1990.

Table V: Frequency and Duration of Employment Related to Possible Mirex Exposure Among 200 Survey Respondents

type of employment	number of respondents	percentage
not related to mirex	189	94.5%
Nease/possible contact	11	5.5%

Table VI: Frequency of Consumption of Products from Animals with Access to MFLBC Among 200 Survey Respondents

consumption frequency	number of respondents	percentage
none	191	95.5%
≤1/year	1	0.5%
1-2/6 months	0	0.05
1-2/month	2	1.0%
1-2/week	0	0.0%
≥3-4/week	6	3.0%

From: Rouse Martin M, Shelley TL, Mortensen BK. Assessment of Exposure to Mirex Associated with the Nease Chemical Company Superfund Site in Salem, Columbiana County, Ohio. Ohio Department of Health. October 4, 1990.

APPENDIX K

Tables of Hazard Index Values and Cancer Risk Estimates for the Exposed Populations

APPENDIX K

Tables of Hazard Index Values and Cancer Risk Estimates for the Exposed Populations

This appendix presents the hazard index and cancer risk tables for the exposed populations. As stated in Chapter VIII of the EA, it is important to understand that the risk values estimated in this assessment are not actuarial risks; that is, they are not risks that have been specifically documented as a result of human exposure to the chemicals retained in the EA. Risk estimates are based on a series of conservative assumptions and, as such, represent an upper bound on risk.

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HAZARD INDEX AND CANCER RISK FOR CURRENT ON-SITE TRESPASSERS EXPOSED TO SOIL

Chemical	Soil Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of On-Site Soil: Current Trespasser	
				Soil Ingestion Trespasser HAZARD INDEX	Soil Ingestion Trespasser CANCER RISK
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	0.0266	NA	2.0E-01	NA	1.0E-10
Benzene	0.002	NA	2.9E-02	NA	1.1E-12
Tetrachloroethene	0.1032	1.0E-02	5.2E-02	1.6E-06	1.1E-10
Trichloroethene	0.007	6.0E-03	1.1E-02	1.8E-07	1.5E-12
SEMIVOLATILE COMPOUNDS					
Hexachlorobenzene	1.5	8.0E-04	1.6E+00	2.9E-04	4.7E-08
Hexachlorobutadiene	0.29	2.0E-04	7.8E-02	2.2E-04	4.4E-10
Hexachloroethane	0.18	1.0E-03	1.4E-02	2.8E-05	5.0E-11
PESTICIDES					
4,4'-DDT	NA	5.0E-04	3.4E-01	NA	NA
OTHER COMPOUNDS					
Mirex	92.8	2.0E-04	5.3E-01	7.1E-02	9.7E-07
				7.1E-02	1.0E-06

HAZARD INDEX AND CANCER RISK FOR FUTURE ON-SITE TRESPASSERS EXPOSED TO SOIL

Chemical	Soil Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	*Ingestion of On-Site Soil: Future Trespasser* Soil Ingestion Trespasser HAZARD INDEX	Soil Ingestion Trespasser CANCER RISK
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	8.2249	NA	2.0E-01	NA	3.2E-08
Benzene	2.4289	NA	2.9E-02	NA	1.4E-09
Tetrachloroethene	19.5207	1.0E-02	5.2E-02	3.0E-04	2.0E-08
Trichloroethene	2.3857	6.0E-03	1.1E-02	6.1E-05	5.2E-10
SEMIVOLATILE COMPOUNDS					
Hexachlorobenzene	2.9593	8.0E-04	1.6E+00	5.7E-04	9.3E-08
Hexachlorobutadiene	0.81	2.0E-04	7.8E-02	6.2E-04	1.2E-09
Hexachloroethane	2.4	1.0E-03	1.4E-02	3.7E-04	6.6E-10
PESTICIDES					
4,4'-DDT	1.0127	5.0E-04	3.4E-01	3.1E-04	6.8E-09
OTHER COMPOUNDS					
Mirex	688.2738	2.0E-04	5.3E-01	5.3E-01	7.2E-06
				5.3E-01	7.3E-06

HAZARD INDEX AND CANCER RISK FOR ON-SITE TRESPASSERS EXPOSED TO AIR

Chemical	Air Conc. (mg/m^3)	RfD (mg/kg-d)	Slope Factor 1/(mg/kg-d)	Inhalation of Air: Current & Future Trespasser	
				Air Inhalation Trespasser HAZARD INDEX	Air Inhalation Trespasser CANCER RISK
VOLATILE COMPOUNDS					
Carbon Tetrachloride	1.67E-05	7.0E-04	5.3E-02	1.2E-04	5.8E-10
SEMIVOLATILE COMPOUNDS					
bis(2-Ethylhexyl)Phthalate	2.55E-02	2.0E-02	1.4E-02	6.5E-03	2.3E-07
N-Nitrosodiphenylamine	2.20E-03	NA	4.9E-03	NA	7.0E-09
OTHER COMPOUNDS					
Mirex	4.70E-05	2.0E-04	5.3E-01	1.2E-03	1.6E-08
				7.8E-03	2.6E-07

HAZARD INDEX AND CANCER RISK FOR TRESPASSERS EXPOSED TO SURFACE WATER

Chemical	Surface Water Conc. (mg/l)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of On-Site Surface Water: Current & Future Trespasser	
				Surface Water Ingestion Trespasser HAZARD INDEX	Surface Water Ingestion Trespasser CANCER RISK
VOLATILE COMPOUNDS					
1,1,2-Trichloroethane	0.0039	4.0E-03	5.7E-02	1.5E-05	4.4E-10
1,1,2,2-Tetrachloroethane	0.47	NA	2.0E-01	NA	1.8E-07
1,2-Dichloroethene (total)	0.014	9.0E-03	NA	2.4E-05	NA
Acetone	0.17	1.0E-01	NA	2.6E-05	NA
Benzene	0.14	NA	2.9E-02	NA	8.0E-09
Carbon Tetrachloride	0.002	7.0E-04	1.3E-01	4.4E-05	5.1E-10
Tetrachloroethene	0.34	1.0E-02	5.2E-02	5.2E-04	3.5E-08
Trichloroethene	0.094	NA	1.1E-02	NA	2.0E-09
SEMIVOLATILE COMPOUNDS					
1,2-Dichlorobenzene	0.18	9.0E-02	NA	3.1E-05	NA
2,4-Dichlorophenol	0.004	3.0E-03	NA	2.0E-05	NA
Hexachloroethane	0.005	1.0E-03	1.4E-02	7.6E-05	1.4E-10
OTHER COMPOUNDS					
Mirex	0.0004	2.0E-04	5.3E-01	3.1E-05	4.2E-10
				7.9E-04	2.3E-07

HAZARD INDEX AND CANCER RISK FOR ON-SITE TRESPASSERS EXPOSED TO SEDIMENT

Chemical	Sediment Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of On-Site Sediment: Current & Future Trespasser — Sediment Ingestion Trespasser HAZARD INDEX	Ingestion of On-Site Sediment: Current & Future Trespasser — Sediment Ingestion Trespasser CANCER RISK
SEMIVOLATILE COMPOUNDS					
Benzo(a)Pyrene	0.31	NA	7.3E+00	NA	1.1E-08
Hexachlorobenzene	3	8.0E-04	1.6E+00	1.4E-04	2.4E-08
OTHER COMPOUNDS					
Mirex	129	2.0E-04	5.3E-01	2.5E-02	3.4E-07
				2.5E-02	3.7E-07

HAZARD INDEX AND CANCER RISK FOR WORKERS ADJACENT TO SITE EXPOSED TO SOIL

Chemical	Soil Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Adjacent Soil: Current & Future Worker — Soil Ingestion Worker HAZARD INDEX	Ingestion of Adjacent Soil: Current & Future Worker — Soil Ingestion Worker CANCER RISK
SEMIVOLATILE COMPOUNDS					
Benzo(a)Pyrene	0.034	NA	7.3E+00	NA	4.3E-08
Benzo(b)Fluoranthene	0.036	NA	7.3E-01	NA	4.6E-09
Benzo(k)Fluoranthene	0.03	NA	7.3E-01	NA	3.8E-09
Pyrene	0.066	3.0E-02	NA	1.1E-06	NA
OTHER COMPOUNDS					
Mirex	0.1	2.0E-04	5.3E-01	2.4E-04	9.3E-09
				2.5E-04	6.1E-08

HAZARD INDEX AND CANCER RISK FOR RESIDENTS ADJACENT TO SITE EXPOSED TO SOIL

Chemical	Soil Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Adjacent Soil: Current & Future Resident				
				Soil Ingestion Adult HAZARD INDEX	Soil Ingestion Child HAZARD INDEX	Soil Ingestion Adult CANCER RISK	Soil Ingestion Child CANCER RISK	Soil Ingestion TOTAL CANCER RISK
SEMIVOLATILE COMPOUNDS								
Benzo(a)Anthracene	0.064	NA	7.3E-01	NA	NA	2.2E-08	5.1E-08	7.3E-08
Benzo(a)Pyrene	0.053	NA	7.3E+00	NA	NA	1.8E-07	4.2E-07	6.1E-07
Benzo(b)Fluoranthene	0.098	NA	7.3E-01	NA	NA	3.4E-08	7.8E-08	1.1E-07
Benzo(k)Fluoranthene	0.098	NA	7.3E-01	NA	NA	3.4E-08	7.8E-08	1.1E-07
bis(2-Ethylhexyl)Phthalate	0.4979	2.0E-02	1.4E-02	3.4E-05	3.2E-04	3.3E-09	7.6E-09	1.1E-08
Indeno(1,2,3-cd)Pyrene	0.047	NA	7.3E-01	NA	NA	1.6E-08	3.8E-08	5.4E-08
Pyrene	0.088	3.0E-02	NA	4.0E-06	3.8E-05	NA	NA	NA
PESTICIDES								
4,4'-DDT	0.0332	5.0E-04	3.4E-01	9.1E-05	8.5E-04	5.3E-09	1.2E-08	1.8E-08
Dieldrin	0.0062	5.0E-05	1.6E+01	1.7E-04	1.6E-03	4.7E-08	1.1E-07	1.6E-07
OTHER COMPOUNDS								
Mirex	0.6393	2.0E-04	5.3E-01	4.4E-03	4.1E-02	1.6E-07	3.7E-07	5.3E-07
				4.7E-03	4.4E-02	5.0E-07	1.2E-06	1.7E-06

HAZARD INDEX AND CANCER RISK FOR RESIDENTS ADJACENT TO SITE EXPOSED TO VEGETABLES

Chemical	Vegetable Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Vegetables Grown on Adjacent Soil: Current & Future Resident — Vegetable Ingestion Adult HAZARD INDEX	Vegetable Ingestion Adult CANCER RISK
SEMIVOLATILE COMPOUNDS					
Benzo(a)Anthracene	0.00006	NA	7.3E-01	NA	1.1E-08
Benzo(a)Pyrene	0.000049	NA	7.3E+00	NA	8.8E-08
Benzo(b)Fluoranthene	0.000091	NA	7.3E-01	NA	1.6E-08
Benzo(k)Fluoranthene	0.000091	NA	7.3E-01	NA	1.6E-08
bis(2-Ethylhexyl)Phthalate	0.00046	2.0E-02	1.4E-02	1.3E-05	1.6E-09
Indeno(1,2,3-cd)Pyrene	0.000044	NA	7.3E-01	NA	7.9E-09
Pyrene	0.000082	3.0E-02	NA	1.6E-06	NA
PESTICIDES					
4,4'-DDT	0.000031	5.0E-04	3.4E-01	3.6E-05	2.6E-09
Dieldrin	5.8E-06	5.0E-05	1.6E+01	6.6E-05	2.3E-08
OTHER COMPOUNDS					
Mirex	0.00059	2.0E-04	5.3E-01	1.7E-03	7.7E-08
				1.8E-03	2.4E-07

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO SURFACE WATER -- CURRENT USE

Chemical	Surface Water Conc. (mg/l)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Surface Water: Current Recreational Visitor	
				Surface Water Ingestion Recreator HAZARD INDEX	Surface Water Ingestion Recreator CANCER RISK
VOLATILE COMPOUNDS					
Chloromethane	0.003	NA	1.3E-02	NA	1.1E-09
SEMIVOLATILE COMPOUNDS					
bis(2-Ethylhexyl)Phthalate	0.006	2.0E-02	1.4E-02	2.1E-05	2.5E-09
				2.1E-05	3.6E-09

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO SEDIMENT -- UPSTREAM OF ADVISORY (CURRENT USE)

Chemical	Sediment Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Sediment: Current Recreational Visitor				
				Sediment Ingestion Adult HAZARD INDEX	Sediment Ingestion Child HAZARD INDEX	Sediment Ingestion Adult CANCER RISK	Sediment Ingestion Child CANCER RISK	Sediment Ingestion TOTAL CANCER RISK
SEMIVOLATILE COMPOUNDS								
4-Methylphenol	0.5577	5.0E-03	NA	1.5E-05	2.4E-04	NA	NA	NA
Benzo(a)Anthracene	0.1	NA	7.3E-01	NA	NA	3.4E-09	1.4E-08	1.7E-08
Benzo(a)Pyrene	0.085	NA	7.3E+00	NA	NA	2.9E-08	1.2E-07	1.5E-07
Benzo(b)Fluoranthene	0.173	NA	7.3E-01	NA	NA	5.9E-09	2.4E-08	3.0E-08
Benzo(k)Fluoranthene	0.173	NA	7.3E-01	NA	NA	5.9E-09	2.4E-08	3.0E-08
Indeno(1,2,3-cd)Pyrene	0.071	NA	7.3E-01	NA	NA	2.4E-09	9.7E-09	1.2E-08
PESTICIDES								
4,4'-DDT	0.191	5.0E-04	3.4E-01	5.2E-05	8.4E-04	3.1E-09	1.2E-08	1.5E-08
OTHER COMPOUNDS								
Mirex	0.497	2.0E-04	5.3E-01	3.4E-04	5.4E-03	1.2E-08	4.9E-08	6.2E-08
				4.1E-04	6.5E-03	6.2E-08	2.5E-07	3.1E-07

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO SEDIMENT -- DOWNSTREAM OF ADVISORY (CURRENT & FUTURE USE)

Chemical	Sediment Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Sediment: Recreational Visitor				
				Sediment Ingestion Adult HAZARD INDEX	Sediment Ingestion Child HAZARD INDEX	Sediment Ingestion Adult CANCER RISK	Sediment Ingestion Child CANCER RISK	Sediment Ingestion TOTAL CANCER RISK
SEMIVOLATILE COMPOUNDS								
4-Methylphenol	0.9501	5.0E-03	NA	5.2E-05	8.7E-04	NA	NA	NA
Benzo(a)Anthracene	0.4	NA	7.3E-01	NA	NA	2.7E-08	1.1E-07	1.4E-07
Benzo(a)Pyrene	0.31	NA	7.3E+00	NA	NA	2.1E-07	8.9E-07	1.1E-06
Benzo(b)Fluoranthene	0.3892	NA	7.3E-01	NA	NA	2.7E-08	1.1E-07	1.4E-07
Benzo(k)Fluoranthene	0.3892	NA	7.3E-01	NA	NA	2.7E-08	1.1E-07	1.4E-07
Indeno(1,2,3-cd)Pyrene	0.15	NA	7.3E-01	NA	NA	1.0E-08	4.3E-08	5.3E-08
PESTICIDES								
4,4'-DDT	NA	5.0E-04	3.4E-01	NA	NA	NA	NA	NA
OTHER COMPOUNDS								
Mirex	0.0124	2.0E-04	5.3E-01	1.7E-05	2.8E-04	6.2E-10	2.6E-09	3.2E-09
				6.9E-05	1.2E-03	3.0E-07	1.3E-06	1.6E-06

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO FISH -- UPSTREAM OF ADVISORY (CURRENT USE)

Chemical	Fish Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Fish: Current Recreational Visitor	
				Fish Ingestion Recreator HAZARD INDEX	Fish Ingestion Recreator CANCER RISK
OTHER COMPOUNDS					
Mirex	1.0422	2.0E-04	5.3E-01	1.2E-01	5.6E-06
Photomirex	0.018	1.3E-03	NA	3.4E-04	NA
				1.2E-01	5.6E-06

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO FISH -- DOWNSTREAM OF ADVISORY (CURRENT & FUTURE USE)

Chemical	Fish Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Fish: Recreational Visitor Fish Ingestion Recreator HAZARD INDEX	 Fish Ingestion Recreator CANCER RISK
OTHER COMPOUNDS					
Mirex	0.0394	2.0E-04	5.3E-01	6.0E-02	2.7E-06
Photomirex	0.0031	1.3E-03	NA	7.6E-04	NA
				6.1E-02	2.7E-06

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO GAME

Chemical	Game Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Game: Current & Future Recreational Visitor — Game Ingestion Recreator HAZARD INDEX	Ingestion of Game: Current & Future Recreational Visitor — Game Ingestion Recreator CANCER RISK
OTHER COMPOUNDS					
Mirex	0.003	2.0E-04	5.3E-01	1.1E-03	4.8E-08
Photomirex	NA	1.3E-03	NA	NA	NA
				1.1E-03	4.8E-08

HAZARD INDEX AND CANCER RISK FOR RESIDENTS ALONG THE MFLBC EXPOSED TO SOIL

Chemical	Soil Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Flood Plain Soil: Current & Future Resident				
				Soil Ingestion Adult HAZARD INDEX	Soil Ingestion Child HAZARD INDEX	Soil Ingestion Adult CANCER RISK	Soil Ingestion Child CANCER RISK	Soil Ingestion TOTAL CANCER RISK
OTHER COMPOUNDS								
Mirex	4.1088	2.0E-04	5.3E-01	2.8E-02	2.6E-01	1.0E-06	2.4E-06	3.4E-06
Photomirex	0.0272	1.3E-03	NA	3.0E-05	2.8E-04	NA	NA	NA
				2.8E-02	2.6E-01	1.0E-06	2.4E-06	3.4E-06

HAZARD INDEX AND CANCER RISK FOR RESIDENTS ALONG THE MFLBC EXPOSED TO VEGETABLES

Chemical	Vegetable Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Vegetables From MFLBC Flood Plain Soil: Current & Future Resident	
				Vegetable Ingestion Adult HAZARD INDEX	Vegetable Ingestion Adult CANCER RISK
OTHER COMPOUNDS					
Mirex	0.0038	2.0E-04	5.3E-01	1.1E-02	4.9E-07
Photomirex	0.000025	1.3E-03	NA	1.1E-05	NA
				1.1E-02	4.9E-07

HAZARD INDEX AND CANCER RISK FOR RESIDENTS ALONG THE MFLBC EXPOSED TO BEEF

Chemical	Beef Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Beef Raised in MFLBC Flood Plain Soil: Future Resident	
				Beef Ingestion Adult HAZARD INDEX	Beef Ingestion Adult CANCER RISK
OTHER COMPOUNDS					
Mirex	0.44	2.0E-04	5.3E-01	1.3E+00	6.0E-05
Photomirex	0.003	1.3E-03	NA	1.4E-03	NA
				1.3E+00	6.0E-05

HAZARD INDEX AND CANCER RISK FOR RESIDENTS ALONG THE MFLBC EXPOSED TO MILK

Chemical	Milk Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Milk From Cows Raised in MFLBC Flood Plain Soil: Future Resident				
				Milk Ingestion Adult HAZARD INDEX	Milk Ingestion Child HAZARD INDEX	Milk Ingestion Adult CANCER RISK	Milk Ingestion Child CANCER RISK	Milk Ingestion TOTAL CANCER RISK
OTHER COMPOUNDS								
Mirex	0.005	2.0E-04	5.3E-01	4.0E-02	3.1E-01	1.5E-06	2.8E-06	4.3E-06
Photomirex	0.00003	1.3E-03	NA	4.2E-05	3.2E-04	NA	NA	NA
				4.0E-02	3.1E-01	1.5E-06	2.8E-06	4.3E-06

HAZARD INDEX AND CANCER RISK FOR FUTURE ON-SITE WORKERS EXPOSED TO GROUND WATER

Chemical	Ground Water Conc. (mg/l)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of On-Site Ground Water: Future Worker	
				Ground Water Ingestion Adult HAZARD INDEX	Ground Water Ingestion Adult CANCER RISK
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	13	NA	2.0E-01	NA	9.1E-03
1,2-Dichloroethane	1	NA	9.1E-02	NA	3.2E-04
1,2-Dichloroethene (total)	15.4	9.0E-03	NA	1.7E+01	NA
Benzene	5.1	NA	2.9E-02	NA	5.2E-04
Chlorobenzene	0.57	2.0E-02	NA	2.8E-01	NA
Tetrachloroethene	100	1.0E-02	5.2E-02	9.8E+01	1.8E-02
Trichloroethene	19	NA	1.1E-02	NA	7.3E-04
SEMIVOLATILE COMPOUNDS					
1,2-Dichlorobenzene	31	9.0E-02	NA	3.4E+00	NA
2,4-Dichlorophenol	0.047	3.0E-03	NA	1.5E-01	NA
Hexachlorobutadiene	0.11	2.0E-04	7.8E-02	5.4E+00	3.0E-05
Hexachloroethane	0.47	1.0E-03	1.4E-02	4.6E+00	2.3E-05
OTHER COMPOUNDS					
Mirex	0.2396	2.0E-04	5.3E-01	1.2E+01	4.4E-04
INORGANIC COMPOUNDS					
Arsenic	0.058	3.0E-04	1.8E+00	1.9E+00	3.6E-04
Beryllium	0.078	5.0E-03	4.3E+00	1.5E-01	1.2E-03
Cadmium	0.123	5.0E-04	NA	2.4E+00	NA
				1.4E+02	3.1E-02

HAZARD INDEX AND CANCER RISK FOR FUTURE ON-SITE WORKERS EXPOSED TO SOIL

Chemical	Soil Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of On-Site Soil: Future Worker	
				Soil Ingestion Worker HAZARD INDEX	Soil Ingestion Worker CANCER RISK
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	8.2249	NA	2.0E-01	NA	6.3E-08
Benzene	2.4289	NA	2.9E-02	NA	1.7E-09
Tetrachloroethene	19.5207	1.0E-02	5.2E-02	3.2E-04	5.9E-08
Trichloroethene	2.3857	6.0E-03	1.1E-02	3.6E-05	8.6E-10
SEMIVOLATILE COMPOUNDS					
Hexachlorobenzene	2.9593	8.0E-04	1.6E+00	1.8E-03	8.3E-07
Hexachlorobutadiene	0.81	2.0E-04	7.8E-02	1.3E-02	7.5E-08
Hexachloroethane	2.4	1.0E-03	1.4E-02	1.9E-03	9.4E-09
PESTICIDES					
4,4'-DDT	1.0127	5.0E-04	3.4E-01	9.8E-04	5.9E-08
OTHER COMPOUNDS					
Mirex	688.2738	2.0E-04	5.3E-01	1.7E+00	6.4E-05
				1.7E+00	6.5E-05

HAZARD INDEX AND CANCER RISK FOR FUTURE ON-SITE WORKERS EXPOSED TO AIR

Chemical	Air Conc. (mg/m^3)	RfD (mg/kg-d)	Slope Factor 1/(mg/kg-d)	Inhalation of Air: Future Worker	
				Air Inhalation Worker HAZARD INDEX	Air Inhalation Worker CANCER RISK
VOLATILE COMPOUNDS					
Carbon Tetrachloride	1.67E-05	7.0E-04	5.3E-02	4.7E-03	6.2E-08
SEMIVOLATILE COMPOUNDS					
bis(2-Ethylhexyl)Phthalate	2.55E-02	2.0E-02	1.4E-02	2.5E-01	2.5E-05
N-Nitrosodiphenylamine	2.20E-03	NA	4.9E-03	NA	7.5E-07
OTHER COMPOUNDS					
Mirex	4.70E-05	2.0E-04	5.3E-01	4.6E-02	1.7E-06
				3.0E-01	2.8E-05

HAZARD INDEX AND CANCER RISK FOR FUTURE ON-SITE RESIDENTS EXPOSED TO GROUND WATER

Chemical	Ground Water Conc. (mg/l)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of On-Site Ground Water: Future Resident	
				Ground Water Ingestion Adult HAZARD INDEX	Ground Water Ingestion Adult CANCER RISK
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	13	NA	2.0E-01	NA	3.1E-02
1,2-Dichloroethane	1	NA	9.1E-02	NA	1.1E-03
1,2-Dichloroethene (total)	15.4	9.0E-03	NA	4.7E+01	NA
Benzene	5.1	NA	2.9E-02	NA	1.7E-03
Chlorobenzene	0.57	2.0E-02	NA	7.8E-01	NA
Tetrachloroethene	100	1.0E-02	5.2E-02	2.7E+02	6.1E-02
Trichloroethene	19	NA	1.1E-02	NA	2.5E-03
SEMIVOLATILE COMPOUNDS					
1,2-Dichlorobenzene	31	9.0E-02	NA	9.4E+00	NA
2,4-Dichlorophenol	0.047	3.0E-03	NA	4.3E-01	NA
Hexachlorobutadiene	0.11	2.0E-04	7.8E-02	1.5E+01	1.0E-04
Hexachloroethane	0.47	1.0E-03	1.4E-02	1.3E+01	7.7E-05
OTHER COMPOUNDS					
Mirex	0.2396	2.0E-04	5.3E-01	3.3E+01	1.5E-03
INORGANIC COMPOUNDS					
Arsenic	0.058	3.0E-04	1.8E+00	5.3E+00	1.2E-03
Beryllium	0.078	5.0E-03	4.3E+00	4.3E-01	3.9E-03
Cadmium	0.123	5.0E-04	NA	6.7E+00	NA
				4.0E+02	1.0E-01

HAZARD INDEX AND CANCER RISK FOR FUTURE ON-SITE RESIDENTS EXPOSED TO SOIL

Chemical	Soil Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of On-Site Soil: Future Resident				
				Soil Ingestion Adult HAZARD INDEX	Soil Ingestion Child HAZARD INDEX	Soil Ingestion Adult CANCER RISK	Soil Ingestion Child CANCER RISK	Soil Ingestion TOTAL CANCER RISK
VOLATILE COMPOUNDS								
1,1,2,2-Tetrachloroethane	8.2249	NA	2.0E-01	NA	NA	7.7E-07	1.8E-06	2.6E-06
Benzene	2.4289	NA	2.9E-02	NA	NA	3.3E-08	7.7E-08	1.1E-07
Tetrachloroethene	19.5207	1.0E-02	5.2E-02	2.7E-03	2.5E-02	4.8E-07	1.1E-06	1.6E-06
Trichloroethene	2.3857	NA	1.1E-02	NA	NA	1.2E-08	2.9E-08	4.1E-08
SEMIVOLATILE COMPOUNDS								
Hexachlorobenzene	2.9593	8.0E-04	1.6E+00	5.1E-03	4.7E-02	2.2E-06	5.2E-06	7.4E-06
Hexachlorobutadiene	0.81	2.0E-04	7.8E-02	5.5E-03	5.2E-02	3.0E-08	6.9E-08	9.9E-08
Hexachloroethane	2.4	1.0E-03	1.4E-02	3.3E-03	3.1E-02	1.6E-08	3.7E-08	5.3E-08
PESTICIDES								
4,4'-DDT	1.0127	5.0E-04	3.4E-01	2.8E-03	2.6E-02	1.6E-07	3.8E-07	5.4E-07
OTHER COMPOUNDS								
Mirex	688.2738	2.0E-04	5.3E-01	4.7E+00	4.4E+01	1.7E-04	4.0E-04	5.7E-04
				4.7E+00	4.4E+01	1.8E-04	4.1E-04	5.8E-04

HAZARD INDEX AND CANCER RISK FOR FUTURE ON-SITE RESIDENTS EXPOSED TO AIR

Chemical	Air Conc. (mg/m^3)	RfD (mg/kg-d)	Slope Factor 1/(mg/kg-d)	Inhalation of Air: Future Resident — Air Inhalation Adult HAZARD INDEX	Inhalation of Air: Future Resident — Air Inhalation Adult CANCER RISK
VOLATILE COMPOUNDS					
Carbon Tetrachloride	1.67E-05	7.0E-04	5.3E-02	6.5E-03	1.0E-07
SEMIVOLATILE COMPOUNDS					
bis(2-Ethylhexyl)Phthalate	2.55E-02	2.0E-02	1.4E-02	3.5E-01	4.2E-05
N-Nitrosodiphenylamine	2.20E-03	NA	4.9E-03	NA	1.3E-06
OTHER COMPOUNDS					
Mirex	4.70E-05	2.0E-04	5.3E-01	6.4E-02	2.9E-06
				4.2E-01	4.6E-05

HAZARD INDEX AND CANCER RISK FOR ON-SITE RESIDENTS EXPOSED TO VEGETABLES

Chemical	Vegetables Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of Vegetables Grown On-Site : Future Resident Vegetables Ingestion Adult HAZARD INDEX	 Vegetables Ingestion Adult CANCER RISK
VOLATILE COMPOUNDS					
1,1,2,2-Tetrachloroethane	0.0076	NA	2.0E-01	NA	3.7E-07
Benzene	0.0023	NA	2.9E-02	NA	1.6E-08
Tetrachloroethene	0.018	1.0E-02	5.2E-02	1.0E-03	2.3E-07
Trichloroethene	0.0022	NA	1.1E-02	NA	5.9E-09
SEMIVOLATILE COMPOUNDS					
Hexachlorobenzene	0.0028	8.0E-04	1.6E+00	2.0E-03	1.1E-06
Hexachlorobutadiene	0.00075	2.0E-04	7.8E-02	2.1E-03	1.4E-08
Hexachloroethane	0.0022	1.0E-03	1.4E-02	1.3E-03	7.6E-09
PESTICIDES					
4,4'-DDT	0.00094	5.0E-04	3.4E-01	1.1E-03	7.8E-08
OTHER COMPOUNDS					
Mirex	0.64	2.0E-04	5.3E-01	1.8E+00	8.3E-05
				1.8E+00	8.5E-05

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO SURFACE WATER -- FUTURE USE

Chemical	Surface Water Conc. (mg/l)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Surface Water: Future Recreational Visitor	
				Surface Water Ingestion Recreator HAZARD INDEX	Surface Water Ingestion Recreator CANCER RISK
VOLATILE COMPOUNDS					
Chloromethane	0.003	NA	1.3E-02	NA	2.3E-09
SEMIVOLATILE COMPOUNDS					
bis(2-Ethylhexyl)Phthalate	0.006	2.0E-02	1.4E-02	4.1E-05	4.9E-09
				4.1E-05	7.2E-09

HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO SEDIMENT -- UPSTREAM OF ADVISORY (FUTURE USE)

Chemical	Sediment Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Sediment: Future Recreational Visitor				
				Sediment Ingestion Adult HAZARD INDEX	Sediment Ingestion Child HAZARD INDEX	Sediment Ingestion Adult CANCER RISK	Sediment Ingestion Child CANCER RISK	Sediment Ingestion TOTAL CANCER RISK
SEMIVOLATILE COMPOUNDS								
4-Methylphenol	0.5577	5.0E-03	NA	3.1E-05	5.1E-04	NA	NA	NA
Benzo(a)Anthracene	0.1	NA	7.3E-01	NA	NA	6.9E-09	2.9E-08	3.5E-08
Benzo(a)Pyrene	0.085	NA	7.3E+00	NA	NA	5.8E-08	2.4E-07	3.0E-07
Benzo(b)Fluoranthene	0.173	NA	7.3E-01	NA	NA	1.2E-08	4.9E-08	6.1E-08
Benzo(k)Fluoranthene	0.173	NA	7.3E-01	NA	NA	1.2E-08	4.9E-08	6.1E-08
Indeno(1,2,3-cd)Pyrene	0.071	NA	7.3E-01	NA	NA	4.9E-09	2.0E-08	2.5E-08
PESTICIDES								
4,4'-DDT	0.191	5.0E-04	3.4E-01	1.0E-04	1.7E-03	6.1E-09	2.5E-08	3.2E-08
OTHER COMPOUNDS								
Mirex	0.497	2.0E-04	5.3E-01	6.8E-04	1.1E-02	2.5E-08	1.0E-07	1.3E-07
				8.2E-04	1.4E-02	1.2E-07	5.2E-07	6.4E-07

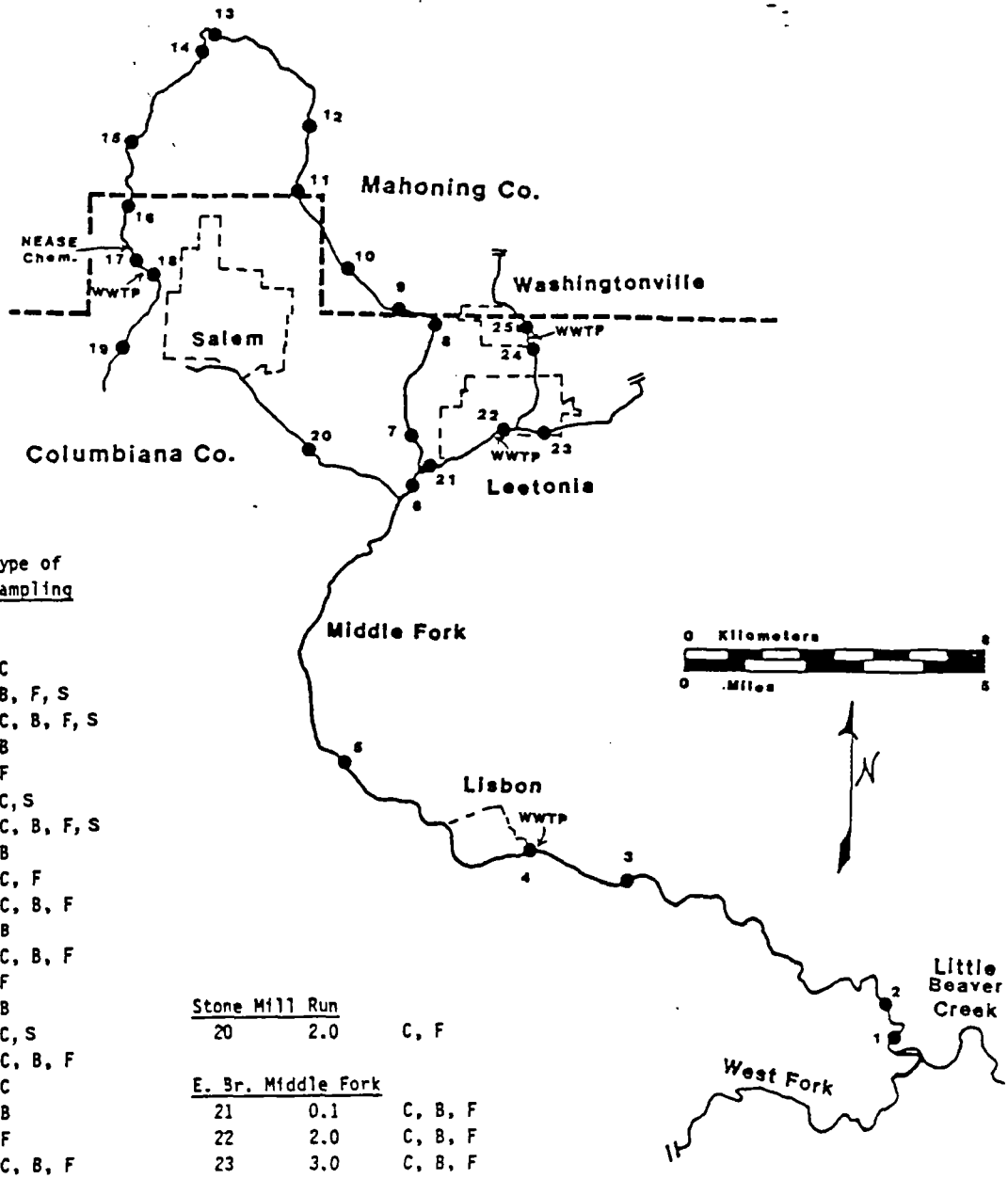
HAZARD INDEX AND CANCER RISK FOR RECREATIONAL VISITORS EXPOSED TO FISH -- UPSTREAM OF ADVISORY (FUTURE USE)

Chemical	Fish Conc. (mg/kg)	Oral RfD (mg/kg-day)	Oral Slope Factor 1/(mg/kg-day)	Ingestion of MFLBC Fish: Future Recreational Visitor	
				Fish Ingestion Recreator HAZARD INDEX	Fish Ingestion Recreator CANCER RISK
OTHER COMPOUNDS					
Mirex	1.0422	2.0E-04	5.3E-01	1.6E+00	7.2E-05
Photomirex	0.018	1.3E-03	NA	4.4E-03	NA
				1.6E+00	7.2E-05

APPENDIX L

Ohio EPA 1985 Biological Survey Information and Related Summaries

Appendix B Figure 2. The location (by river mile) of water chemistry (C), fish (F), benthic (B) and sediment (S) sampling sites in the Middle Fork Little Beaver Creek study area during JUNE ~~July~~–October, 1985.



Site Number	River Mile	Type of Sampling
Middle Fork		
1	0.5	C
2	1.9	B, F, S
3	9.0	C, B, F, S
4	10.6	B
	10.9	F
	11.0	C, S
5	15.1	C, B, F, S
6	20.8	B
	20.9	C, F
7	21.8	C, B, F
8	24.8	B
9	25.1	C, B, F
10	26.8	F
	26.9	B
11	27.8	C, S
12	28.8	C, B, F
13	32.0	C
14	32.6	B
	32.7	F
15	35.4	C, B, F
16	36.7	C, B, F, S
17	37.6	F
	37.7	C, B
18	38.3	C, B, F
19	40.3	C, B, F
Stone Mill Run		
20	2.0	C, F
E. Br. Middle Fork		
21	0.1	C, B, F
22	2.0	C, B, F
23	3.0	C, B, F
Cherry Valley Run		
24	2.0	B, F
	2.1	C
25	2.4	C

INTER-OFFICE COMMUNICATION

To: Sherry Koslowsky, DE&RR

Date:Mon, Mar 30, 1992

From: Chris Yoder,Manager, EA Section-WQP&A C Yoder

Subject: Middle Fork Little Beaver Creek Information

I transmitted some data and information about the M. Fk. L. Beaver Creek to you some time ago. I just realized that the aquatic life use attainment table you received was erroneously based on the Warmwater Habitat (WWH) use designation. It should have been based on the Exceptional Warmwater Habitat (EWH) use designation. FULL attainment of EWH was not observed at any site in the Middle Fork. It is also interesting to note that the "strength" of the PARTIAL attainment increases downstream to RM 1.9. This indicates that the impacts from the Salem area may extend downstream for nearly 30 miles.

cc: Dave Altfater/Bernie Counts
~~*Biolab Files (08-100)~~

Table 1. Aquatic life use attainment status for the Exceptional Warmwater Habitat (EWH) use designation in Middle Fork L.Beaver Creek based on data collected during July-October 1985 and July-September 1987, and for the Warmwater Habitat (WWH) use designation in the Nease Tributary in August 1991.

RIVER MILE Fish/Invert.	IBI	Modified Iwb	ICI[a]	QHEI[b]	Attainment Status[c]	Comment
Nease Tributary (1991)						
0.2/ -	38	N/A	--	61.5	(FULL)	Ust. Nease leachate area
Middle Fork L. Beaver Creek (1985)						
40.3/40.3	37*	N/A	18*	59	NON	
38.3/ -	31*	N/A	F*	47	NON	Dst. Buttermilk Cr. discharges
37.6/37.7	24*	N/A	0*	55.5	NON	Dst. Salem WWTP
36.7/36.7	25*	N/A	6*	66	NON	Dst. Nease tributary
35.4/35.4	32*	N/A	30*	68	NON	
32.7/32.6	25*	N/A	38	51.5	NON	
28.8/28.8	25*	5.6*	24*	41	NON	
26.8/26.9	27*	5.1*	40	43	NON	
25.1[d]/25.1	27*	4.7*	18*	48	NON	
21.8/21.8	37*	7.1*	28*	55.5	NON	
20.9[d]/20.9	24*	6.3*	38	32	NON	
15.1/15.1	35*	7.7*	50	89	PARTIAL	
10.9/10.9	43*	8.9[ns]	40*	73	PARTIAL	Ust. Lisbon WWTP
9.0/9.0	45*	9.2[ns]	32[ns]	87	PARTIAL	Dst. Lisbon WWTP
1.9/1.9	48[ns]	8.7*	46	81	PARTIAL	Regional reference site
Middle Fork L. Beaver Creek (1987)						
25.1[d]/ -	22*	4.9*	--	-	(NON)	
15.1/ -	38*	8.0*	--	-	(NON)	

* - significant departure from ecoregion biocriteria; poor and very poor results are underlined.

[ns]-nonsignificant departure from ecoregion biocriteria (4 IBI or ICI units; 0.5 Iwb units).

[a] - Narrative criteria used when ICI is not available (G = Good; F = Fair; P = Poor).

[b] - Qualitative Habitat Evaluation Index (QHEI) values are based on the most recent version (Rankin 1989);

[c] - use attainment status based on one organism group is parenthetically expressed.

[d] - boat site; all others are wading site type.

Ecoregion Biocriteria: Erie/Ontario Lake Plain (EOLP)

INDEX - Site Type	WWH	EWH	MWH[d]
IBI - Wading	40	50	24
IBI - Boat	40	48	24
Mod. Iwb - Wading	7.9	9.4	6.2
Mod. Iwb - Boat	8.7	9.6	5.8
ICI	34	46	22

[d] - Modified Warmwater Habitat for channel modified areas.

INTER-OFFICE COMMUNICATION

To: Sherry Koslowsky, DERR Date:Fri, Nov 8, 1991

From: Chris Yoder, Manager, EA Section-WQP&A C Yoder

Subject Monitoring Results For M. Fork L. Beaver Creek

The monitoring results that you requested for M. Fork L. Beaver Creek are attached. Table 1 is a standard aquatic life use attainment table which was constructed utilizing sampling results from 1985, 1987, and 1991. This represents how the ambient biological data is used according to the Ohio WQS (OAC 3745-1). We can provide more detailed summaries of the data if you wish. The use attainment table demonstrates a failure to fully attain the WWH biological criteria from RM 40.3 to RM 20.9. The most severe impact occurred immediately downstream from the Salem WWTP, but it is possible that other sources upstream are contributing to the impairment. We believe that multiple sources are responsible and include small industries that discharge to Buttermilk Creek, the Salem WWTP, and Nease Chemical. We also observed an unusual proportion of deformities (primarily "pug headedness") on fish in the area downstream from Nease Chemical where mirex has been measured in both the sediment and fish tissue. This type of deformity is not observed in streams impacted predominantly by municipal sewage. Habitat has also been disturbed, but is not extensive enough to preclude eventual attainment of the WWH criteria. The average QHEI for the M. Fork is 60 which is sufficient to support the WWH use. I would presume that the Salem WWTP has upgraded their treatment facilities since the 1985 survey. Thus it may be useful to conduct a follow-up survey to better determine the contribution of Nease Chemical to the overall aquatic life impairment. We are not scheduled to revisit this basin until 1994; however, we could use our DERR staff to conduct some of this work earlier. Also, Dave Altfater and Bernie Counts conducted an evaluation (at the request of DERR) of the unnamed tributary that flows through the Nease Chemical site. Their evaluation is attached.

Tables 2 and 3 are the metric scores for the IBI (fish) and ICI (macroinvertebrates) which provides an opportunity to examine the community response "signatures". Although the data strongly suggests sewage enrichment, some of the metrics are also characteristic of toxicity. We could provide a more detailed assessment if you wish.

Table 4 is a summary of the Area of Degradation Value statistics for the M. Fork. ADV is a statistic that is derived from the area formed by a plot of the IBI, ICI, etc. by river mile. A concept paper is attached that explains how the ADV is derived with suggestions for its use. One remaining challenge is to determine an objective method to allocate ADV points among multiple sources of impairment. With regard to the M. Fork the IBI ADV/mile of 93 is comparable to some of the most degraded rivers statewide.

The available water column chemical data is provided on the attached STORET printout. I did not examine the results for criteria exceedences. Also included is sediment metals data from 6 sites. We have been using the Illinois EPA sediment ranking method in lieu of any other type of criteria. These rankings are based on the severity of departure of values above background levels. We are developing a similar system for Ohio based on our sediment database. Fish tissue data is attached as well. The only chemicals that appeared above detectable levels were mirex, chlordane, and PCB-1260. Fish tissue analysis was restricted to PCB and pesticides.

cc: Biolab Files (08-200)
Dave Altfater
Bob Wysenski, NEDO

Table 1. Aquatic life use attainment status for the Warmwater Habitat (WWH) use designation in Middle Fork L.Beaver Creek based on data collected during July-October 1985 and July-September 1987, and the Nease Tributary in August 1991.

RIVER MILE Fish/Invert.	IBI	Modified Iwb	ICI[a]	QHEI[b]	Attainment Status[c]	Comment
Nease Tributary (1991)						
0.2/ -	38	N/A	--	61.5	(FULL)	Ust. Nease leachate area
Middle Fork L. Beaver Creek (1985)						
40.3/40.3	37[ns]	N/A	18*	59	PARTIAL	
38.3/ -	31*	N/A	F	47	NON	Dst. Buttermilk Cr. discharges
37.6/37.7	24*	N/A	0*	55.5	NON	Dst. Salem WWTP
36.7/36.7	25*	N/A	6*	66	NON	Dst. Nease tributary
35.4/35.4	32*	N/A	30[ns]	68	PARTIAL	
32.7/32.6	25*	N/A	38	51.5	NON	
28.8/28.8	25*	5.6*	24*	41	NON	
26.8/26.9	27*	5.1*	40	43	NON	
25.1[d]/25.1	27*	4.7*	18*	48	NON	
21.8/21.8	37[ns]	7.1*	28*	55.5	PARTIAL	
20.9[d]/20.9	24*	6.3*	38	32	NON	
15.1/15.1	35	7.7[ns]	50	89	FULL	
10.9/10.9	43	8.9	40	73	FULL	Ust. Lisbon WWTP
9.0/9.0	45	9.2	32[ns]	87	FULL	Dst. Lisbon WWTP
1.9/1.9	48	8.7	46	81	FULL	Regional reference site
Middle Fork L. Beaver Creek (1987)						
25.1[d]/ -	22*	4.9*	--	-	(NON)	
15.1/ -	38	8.0	--	-	(FULL)	

* - significant departure from ecoregion biocriteria; poor and very poor results are underlined.

[ns]-nonsignificant departure from ecoregion biocriteria (4 IBI or ICI units; 0.5 Iwb units).

[a] - Narrative criteria used when ICI is not available (G = Good; F = Fair; P = Poor).

[b] - Qualitative Habitat Evaluation Index (QHEI) values are based on the most recent version (Rankin 1989);

[c] - use attainment status based on one organism group is parenthetically expressed.

[d] - boat site; all others are wading site type.

Ecoregion Biocriteria: Erie/Ontario Lake Plain (EOLP)

INDEX - Site Type	WWH	EWH	MWH[d]
IBI - Wading	40	50	24
IBI - Boat	40	48	24
Mod. Iwb - Wading	7.9	9.4	6.2
Mod. Iwb - Boat	8.7	9.6	5.8
ICI	34	46	22

[d] - Modified Warmwater Habitat for channel modified areas.

Table 2. IBI table for the M. Fork L. Beaver Creek based on data collected in 1985.

River Mile	Type	Date	Drainage area (sq mi)	Number of: Total species	Minnow species	Headwater species	Sensitive species	Darter & Sculpin species	Simple Lithophils	Percent of Individuals: Tolerant fishes	Omni-vores	Pioneering fishes	Insect-ivores	DELT anomalies	Rel.No. minus tolerants /(0.3km)	IBI
M. FK. L. BEAVER CRK - (08-200)																
Year: 85																
40.3	E	09/05/85	1.7	8(5)	3(3)	2(3)	0(1)	1(3)	2(3)	69(1)	27(1)	54(3)	32(5)	0.0(5)	598(5)	38
40.3	E	08/15/85	1.7	11(5)	6(5)	2(3)	0(1)	1(3)	3(5)	82(1)	27(1)	59(1)	19(3)	0.0(5)	506(5)	38
40.3	E	07/10/85	1.7	9(5)	4(3)	2(3)	0(1)	1(3)	2(3)	74(1)	26(1)	63(1)	27(5)	0.0(5)	482(5)	36
38.3	D	09/05/85	5.0	15(5)	5(3)	2(3)	1(1)	2(3)	3(3)	86(1)	22(3)	58(1)	24(3)	0.2(3)	119(1)	30
38.3	D	08/14/85	5.0	16(5)	6(5)	2(3)	1(1)	2(3)	3(3)	83(1)	32(1)	46(3)	26(3)	0.2(3)	177(3)	34
38.3	E	07/10/85	5.0	15(5)	5(3)	2(3)	1(1)	2(3)	3(3)	91(1)	30(1)	55(3)	21(3)	0.2(3)	63(1)	30
37.6	D	09/04/85	6.0	11(3)	5(3)	2(3)	0(1)	0(1)	2(1)	99(1)	36(1)	59(1)	5(1)	0.7(3)	13(1)	20
37.6	D	08/14/85	6.0	12(3)	5(3)	2(3)	1(1)	1(1)	3(3)	97(1)	62(1)	32(3)	4(1)	1.4(1)	19(1)	22
37.6	D	07/10/85	6.0	14(5)	4(3)	3(3)	1(1)	3(3)	3(3)	96(1)	54(1)	40(3)	7(1)	0.0(5)	27(1)	30
36.7	D	09/04/85	9.0	15(5)	8(5)	3(3)	1(1)	1(1)	3(3)	98(1)	46(1)	47(3)	7(1)	4.7(1)	27(1)	26
36.7	D	08/14/85	9.0	11(3)	5(3)	1(1)	1(1)	2(3)	3(3)	99(1)	53(1)	44(3)	3(1)	0.8(3)	9(1)	24
36.7	D	07/10/85	9.0	14(5)	4(3)	3(3)	0(1)	1(3)	2(1)	99(1)	64(1)	31(3)	4(1)	0.5(3)	10(1)	26
35.4	E	09/04/85	12.0	15(5)	7(5)	5(5)	1(1)	3(3)	4(3)	79(1)	25(3)	50(3)	12(1)	3.5(1)	378(3)	34
35.4	D	08/14/85	12.0	13(3)	8(5)	5(5)	2(1)	2(3)	5(3)	79(1)	34(1)	45(3)	8(1)	0.0(5)	244(3)	34
35.4	E	07/11/85	12.0	15(5)	6(3)	3(3)	1(1)	2(3)	3(1)	81(1)	34(1)	45(3)	9(1)	0.1(3)	322(3)	28
32.7	E	09/04/85	18.0	16(3)	7(5)	1(1)	1(1)	2(1)	4(3)	85(1)	74(1)	63(1)	15(1)	0.5(3)	226(3)	24
32.7	E	08/14/85	18.0	17(5)	6(3)	3(3)	1(1)	2(3)	3(1)	78(1)	54(1)	50(3)	24(1)	1.1(3)	174(1)	26
32.7	D	07/11/85	18.0	13(3)	4(3)	3(3)	1(1)	2(3)	3(1)	79(1)	49(1)	39(3)	24(3)	2.8(1)	124(1)	24

Table 2. IBI table for the M. Fork L. Beaver Creek based on data collected in 1985.

River Mile	Type	Date	Drainage area (sq mi)	Number of: Total species	Sunfish species	Sucker species	Intolerant species	Darter species	Percent of Individuals: Simple Lithophils	Tolerant fishes	Omni-vores	Top carnivores	Insect-ivores	DELT anomalies	Rel.No. minus tolerants /(0.3km)		Modified IBI	Iwb
M. FK. L. BEAVER CRK - (08200)																		
Year: 85																		
28.8	D	09/04/85	26	11(3)	3(3)	1(1)	0(1)	2(3)	40(5)	68(1)	43(1)	8(5)	47(3)	2.7(1)	101(1)		28	5.8
28.8	D	07/15/85	26	14(3)	3(3)	1(1)	0(1)	2(3)	27(3)	73(1)	47(1)	6(5)	44(3)	0.5(3)	85(1)		28	5.4
26.8	D	08/28/85	30	13(3)	3(3)	1(1)	0(1)	2(3)	40(5)	68(1)	36(1)	14(5)	46(3)	2.1(1)	45(1)	*	28	5.3
26.8	D	08/13/85	30	9(1)	2(3)	1(1)	0(1)	1(1)	21(3)	69(1)	21(3)	24(5)	53(3)	0.0(5)	36(1)	*	28	4.9
26.8	D	07/15/85	30	12(3)	2(3)	1(1)	0(1)	3(3)	34(3)	70(1)	56(1)	17(5)	22(1)	0.6(3)	80(1)		26	5.2
21.8	D	08/28/85	41	18(3)	4(5)	2(3)	0(1)	4(3)	35(3)	53(1)	25(3)	12(5)	50(3)	0.5(3)	132(1)		34	7.1
21.8	D	08/13/85	41	16(3)	3(3)	2(3)	0(1)	4(3)	42(5)	47(1)	25(3)	11(5)	54(3)	0.0(5)	113(1)		36	6.9
21.8	D	07/17/85	41	17(3)	4(5)	2(3)	0(1)	5(5)	40(5)	45(3)	33(3)	8(5)	48(3)	0.5(3)	201(3)		42	7.2
15.1	D	08/27/85	96	15(3)	2(3)	2(3)	0(1)	4(3)	10(1)	9(5)	2(5)	0(1)	39(3)	0.4(3)	813(5)		36	7.5
15.1	D	08/13/85	96	14(3)	1(1)	2(3)	0(1)	3(3)	13(1)	13(5)	3(5)	1(1)	42(3)	0.2(3)	565(3)		32	7.6
15.1	D	07/17/85	96	16(3)	1(1)	2(3)	0(1)	5(5)	14(1)	19(5)	8(5)	0(1)	48(3)	0.0(5)	880(5)		38	8.1
10.9	D	08/27/85	105	27(5)	5(5)	4(3)	2(1)	5(3)	41(5)	25(3)	20(3)	6(5)	49(3)	0.4(3)	647(3)		42	9.5
10.9	D	08/12/85	105	26(5)	5(5)	4(3)	2(1)	6(5)	41(5)	24(3)	20(3)	2(3)	48(3)	0.0(5)	618(3)		44	8.7
10.9	D	07/18/85	105	25(5)	3(3)	4(3)	2(1)	6(5)	40(5)	37(3)	28(3)	9(5)	49(3)	1.0(3)	262(3)		42	8.4
9.0	D	08/27/85	114	20(3)	3(3)	2(1)	3(3)	5(3)	51(5)	14(5)	8(5)	1(1)	51(3)	1.5(1)	1346(5)		38	9.3
9.0	D	08/08/85	114	23(5)	2(3)	2(1)	5(3)	7(5)	48(5)	10(5)	5(5)	1(3)	47(3)	0.0(5)	1602(5)		48	8.9
9.0	D	07/18/85	114	23(5)	3(3)	3(3)	4(3)	6(5)	37(5)	7(5)	5(5)	1(3)	38(3)	0.0(5)	2230(5)		50	9.2
1.9	D	08/26/85	141	23(5)	4(5)	3(3)	4(3)	3(3)	32(3)	23(3)	14(5)	14(5)	52(3)	0.6(3)	188(1)		42	8.0
1.9	D	07/18/85	141	28(5)	5(5)	5(5)	4(3)	6(5)	62(5)	22(3)	11(5)	6(5)	79(5)	0.0(5)	303(3)		54	9.4

na - Qualitative da[illegible]dified Iwb not applicable.

Table 2. IBI table for the M. Fork L. Beaver Creek based on data collected in 1985.

River Mile	Type	Date	Drainage area (sq mi)	Number of: Total species	Sunfish species	Sucker species	Intolerant species	Percent of Individuals: Rnd-bodied suckers	Simple Lithophils	Tolerant fishes	Omni-vores	Top carnivores	Insect-ivores	DELT anomalies	Rel.No. minus tolerants /(1.0 km)	Modified IBI	Modified Iwb
M. FK. L. BEAVER CRK - (08-200)																	
Year:	85																
25.1	A	08/28/85	34	8(1)	2(3)	1(1)	0(1)	0(1)	54(5)	61(1)	59(1)	25(5)	16(1)	0.0(5)	54(1) *	26	4.6
25.1	A	08/13/85	34	12(3)	3(3)	2(1)	0(1)	2(1)	49(3)	60(1)	56(1)	12(5)	33(3)	0.0(5)	34(1) *	28	5.7
25.1	A	07/22/85	34	5(1)	2(3)	1(1)	0(1)	0(1)	67(5)	81(1)	81(1)	14(5)	5(1)	0.0(5)	16(1) *	26	3.8
20.9	A	08/28/85	73	16(3)	6(5)	2(1)	0(1)	12(1)	24(1)	61(1)	54(1)	6(3)	36(3)	5.5(1)	126(1)	22	5.9
20.9	A	08/13/85	73	9(1)	2(3)	2(1)	0(1)	7(1)	32(3)	36(1)	36(1)	11(5)	46(3)	0.0(5)	90(1) *	26	6.6

Table 3. ICI table for the M. Fork L. Beaver Creek based on data collected in 1985.

River Mile	Drainage Area (sq mi)	Number of: Total Taxa	Mayfly Taxa	Caddisfly Taxa	Dipteran Taxa	Percent: Mayflies	Caddis-flies	Tany-tarsini	Other Dipt/NI	Tolerant Taxa	Qual. EPT	Eco-region	ICI
M. Fk. L. BEAVER CR. - 08-200													
Year: 85													
40.3	1.7	19(2)	0(0)	0(0)	13(2)	0.0(0)	0.0(0)	29.9(6)	69.8(0)	17.9(4)	7(4)	3	18
37.7	6.0	6(0)	0(0)	0(0)	5(0)	0.0(0)	0.0(0)	0.0(0)	99.9(0)	64.3(0)	0(0)	3	0
36.7	9.0	11(0)	0(0)	0(0)	6(0)	0.0(0)	0.0(0)	18.0(6)	81.9(0)	65.3(0)	1(0)	3	6
35.4	12.0	24(2)	0(0)	3(6)	12(2)	0.0(0)	2.8(6)	48.3(6)	48.7(2)	3.7(6)	2(0)	3	30
32.6	18.0	32(4)	3(2)	4(6)	16(4)	3.3(2)	2.9(4)	56.1(6)	37.5(4)	10.9(4)	6(2)	3	38
28.8	26.0	35(4)	3(2)	1(2)	19(4)	1.1(2)	0.3(2)	25.5(6)	72.9(0)	15.8(2)	3(0)	3	24
26.9	30.0	30(4)	3(2)	2(4)	20(6)	1.0(2)	6.3(4)	57.7(6)	33.7(4)	6.2(6)	4(2)	3	40
25.1	34.0	24(2)	2(0)	1(2)	19(4)	1.9(2)	0.3(2)	26.8(6)	71.0(0)	30.7(0)	3(0)	3	18
21.8	41.0	29(4)	3(2)	6(6)	11(2)	3.1(2)	13.2(6)	5.3(2)	78.0(0)	18.8(2)	6(2)	3	28
20.9	73.0	42(6)	7(4)	5(6)	21(6)	3.2(2)	22.1(6)	16.4(4)	57.0(2)	13.1(2)	4(0)	3	38
15.1	96.0	37(4)	7(4)	4(6)	21(6)	19.1(4)	27.7(6)	31.2(4)	21.5(6)	3.8(6)	10(4)	3	50
10.9	105.0	41(6)	6(4)	6(6)	21(6)	0.9(2)	11.1(4)	43.4(6)	44.4(4)	23.9(0)	8(2)	3	40
9.0	114.0	48(6)	7(4)	2(2)	28(6)	1.1(2)	7.9(2)	30.4(4)	60.0(2)	10.6(2)	6(2)	4	32
1.9	141.0	47(6)	9(6)	4(4)	21(6)	19.7(4)	17.0(4)	20.1(4)	42.4(4)	7.4(4)	11(4)	4	46

Table 21. Relative number of fish (no./0.3km) and total number of species collected by location (river mile - RM) in the Middle Fork Little Beaver Creek during July - September, 1985. Relative numbers at RM 20.9 and 25.1 are per 1.0 km.

SPECIES	RM 1.9	RM 9.0	RM 10.9	RM 15.1	RM 20.9
BOWFIN	-	-	0.5	-	-
GIZZARD SHAD	-	-	3.9	-	2.3
CENTRAL MUDMINNOW	1.5	0.7	-	1.5	-
GRASS PICKEREL	0.8	-	0.5	0.5	3.2
CHAIN PICKEREL	-	-	-	-	-
SILVER REDHORSE	30.8	-	3.0	-	-
BLACK REDHORSE	5.3	-	-	-	-
GOLDEN REDHORSE	10.5	0.7	5.4	-	-
NORTHERN HOG SUCKER	37.5	260.0	169.0	23.0	18.2
WHITE SUCKER	12.0	53.3	45.9	35.0	32.3
COMMON CARP	21.8	0.7	21.2	-	46.5
GOLDEN SHINER	-	-	-	-	-
RIVER CHUB	0.8	0.7	-	-	-
BLACKNOSE DACE	-	-	-	1.0	-
CREEK CHUB	-	74.7	7.4	77.5	4.5
SOUTH. REDBELLY DACE	-	-	-	-	-
REDSIDE DACE	-	-	-	-	-
SILVER SHINER	6.0	53.3	11.6	-	-
ROSYFACE SHINER	0.8	33.3	0.5	-	-
STRIPED SHINER	27.8	86.0	9.2	15.0	1.0
SPOTFIN SHINER	1.5	-	-	-	-
SAND SHINER	0.8	3.3	-	-	-
SILVERJAW MINNOW	-	1.3	1.0	0.5	-
FATHEAD MINNOW	-	-	-	-	-
BLUNTNOSE MINNOW	3.8	55.3	79.1	5.5	19.7
CENTRAL STONEROLLER	28.5	854.7	161.6	373.5	-
CHANNEL CATFISH	0.8	-	0.5	-	-
YELLOW BULLHEAD	3.8	5.3	18.1	1.0	2.3
BROWN BULLHEAD	-	-	2.4	-	5.7
BLACK BULLHEAD	-	-	-	-	2.2
STONECAT MADTOM	-	2.7	-	-	-
WHITE CRAPPIE	-	-	-	-	0.7
BLACK CRAPPIE	1.5	-	1.5	-	0.7
ROCK BASS	13.5	13.3	24.9	2.5	2.5
SMALLMOUTH BASS	8.3	6.7	2.4	-	-
LARGEMOUTH BASS	5.3	0.7	8.8	1.0	10.8
WARMOUTH SF	1.5	-	-	-	0.5
GREEN SUNFISH	27.0	3.3	13.3	1.0	9.7
BLUEGILL SUNFISH	15.0	2.0	47.2	-	29.5
PUMPKINSEED SUNFISH	1.5	0.7	2.4	-	16.3
B'GILL X PUMPKINSEED	-	1.3	-	-	-
GREEN SF X BLUEGILL	-	-	0.5	-	2.2
GR'N SF X PUMPKINS'D	-	2.0	0.5	-	2.8
YELLOW PERCH	-	-	-	-	4.8
BLACKSIDE DARTER	-	-	-	0.5	-
LOGPERCH	3.0	3.3	4.5	-	-
JOHNNY DARTER	3.8	4.7	10.9	6.5	15.3
GREENSIDE DARTER	5.3	168.0	14.9	14.5	4.5
BANDED DARTER	8.3	130.7	12.4	-	-
VARIEGATE DARTER	-	0.7	-	-	-
RAINBOW DARTER	11.3	58.0	7.0	22.0	-
FANTAIL DARTER	0.8	20.7	1.0	57.0	-
FRESHWATER DRUM	3.8	-	1.5	-	-
MOTTLED SCULPIN	11.3	17.3	2.5	236.5	-
BROOK STICKLEBACK	-	-	-	-	-
TOTAL RELATIVE NUMBER	315.0	1919.3	696.8	875.5	238.2
TOTAL NUMBER SPECIES	34.0	30.0	33.0	20.0	22.0
TOTAL NUMBER HYBRIDS	0.0	2.0	2.0	0.0	2.0

Table 21. (Continued).

SPECIES	RM 21.8	RM 25.1	RM 26.8	RM 28.8	RM 32.7
BOWFIN	-	-	-	-	-
GIZZARD SHAD	-	-	-	-	-
CENTRAL MUDMINNOW	0.5	-	12.5	8.3	0.7
GRASS PICKEREL	14.5	10.0	30.5	15.8	-
CHAIN PICKEREL	1.5	0.7	-	6.8	-
SILVER REDHORSE	-	-	-	-	-
BLACK REDHORSE	-	-	-	-	-
GOLDEN REDHORSE	-	-	-	-	-
NORTHERN HOG SUCKER	14.0	0.7	-	-	-
WHITE SUCKER	66.0	56.0	50.0	99.0	262.0
COMMON CARP	0.5	6.0	-	18.0	-
GOLDEN SHINER	-	-	-	3.8	-
RIVER CHUB	-	-	-	-	-
BLACKNOSE DACE	1.5	-	-	-	16.7
CREEK CHUB	18.0	-	5.5	2.3	132.7
SOUTH. REDBELLY DACE	-	-	-	-	-
REDSIDE DACE	-	-	-	-	-
SILVER SHINER	-	-	-	-	-
ROSYFACE SHINER	-	-	-	-	-
STRIPED SHINER	0.5	-	1.0	2.3	1.3
SPOTFIN SHINER	-	-	-	-	-
SAND SHINER	-	-	-	-	-
SILVERJAW MINNOW	-	-	-	-	12.7
FATHEAD MINNOW	-	-	-	0.8	1.3
BLUNTNOSE MINNOW	14.5	4.7	25.0	24.0	337.3
CENTRAL STONEROLLER	14.5	-	-	-	8.7
CHANNEL CATFISH	-	-	-	-	-
YELLOW BULLHEAD	27.0	-	19.5	48.0	21.3
BROWN BULLHEAD	-	2.0	-	-	-
BLACK BULLHEAD	-	0.7	-	-	0.7
STONECAT MADTOM	-	-	-	-	-
WHITE CRAPPIE	-	-	-	-	-
BLACK CRAPPIE	-	0.7	-	-	-
ROCK BASS	12.0	-	-	-	-
SMALLMOUTH BASS	-	-	-	-	-
LARGEMOUTH BASS	-	8.0	0.5	-	2.0
WARMOUTH SF	-	-	-	-	-
GREEN SUNFISH	11.5	-	9.0	16.5	4.0
BLUEGILL SUNFISH	2.5	5.3	4.0	28.5	19.3
PUMPKINSEED SUNFISH	5.5	4.7	1.0	26.3	6.0
B'GILL X PUMPKINSEED	-	-	-	-	-
GREEN SF X BLUEGILL	-	-	0.5	0.8	0.7
GR'N SF X PUMPKINS'D	1.0	-	1.0	3.8	4.0
YELLOW PERCH	-	-	-	-	-
BLACKSIDE DARTER	0.5	-	-	-	-
LOGPERCH	-	-	-	-	-
JOHNNY DARTER	22.5	2.7	7.0	5.3	28.7
GREENSIDE DARTER	22.0	1.3	6.0	3.8	86.0
BANDED DARTER	-	-	-	-	-
VARIEGATE DARTER	-	-	-	-	-
RAINBOW DARTER	7.5	-	-	-	-
FANTAIL DARTER	15.0	-	0.5	-	-
FRESHWATER DRUM	-	-	-	-	-
MOTTLED SCULPIN	15.0	-	1.5	-	1.3
BROOK STICKLEBACK	-	-	-	-	3.3
TOTAL RELATIVE NUMBER	288.0	103.4	175.0	313.5	950.7
TOTAL NUMBER SPECIES	22.0	14.0	15.0	16.0	19.0
TOTAL NUMBER HYBRIDS	1.0	0.0	2.0	2.0	2.0

Table 21. (Continued).

SPECIES	RM 35.4	RM 36.7	RM 37.6	RM 38.3	RM 40.3
BOWFIN	-	-	-	-	-
GIZZARD SHAD	-	-	-	-	-
CENTRAL MUDMINNOW	1.3	4.0	2.5	52.0	4.0
GRASS PICKEREL	-	-	-	-	-
CHAIN PICKEREL	-	-	-	-	-
SILVER REDHORSE	-	-	-	-	-
BLACK REDHORSE	-	-	-	-	-
GOLDEN REDHORSE	-	-	-	-	-
NORTHERN HOG SUCKER	-	-	-	-	-
WHITE SUCKER	314.7	563.0	448.0	220.5	306.0
COMMON CARP	-	-	-	-	-
GOLDEN SHINER	2.7	5.0	20.0	21.0	-
RIVER CHUB	-	-	-	-	-
BLACKNOSE DACE	264.0	13.0	7.5	15.5	454.7
CREEK CHUB	489.3	441.0	453.5	377.5	596.7
SOUTH. REDBELLY DACE	2.0	-	-	-	-
REDSIDE DACE	0.7	1.5	-	-	-
SILVER SHINER	-	-	-	-	-
ROSYFACE SHINER	-	-	-	-	-
STRIPED SHINER	-	-	-	-	0.7
SPOTFIN SHINER	-	-	-	-	-
SAND SHINER	-	-	-	-	-
SILVERJAW MINNOW	25.3	2.0	-	-	-
FATHEAD MINNOW	4.0	3.0	1.0	1.5	0.7
BLUNTNOSE MINNOW	154.7	0.5	1.0	22.5	275.3
CENTRAL STONEROLLER	167.3	1.5	1.0	19.0	4.7
CHANNEL CATFISH	-	-	-	-	-
YELLOW BULLHEAD	2.0	28.0	4.5	7.0	22.0
BROWN BULLHEAD	-	-	1.0	-	-
BLACK BULLHEAD	-	-	-	-	-
STONECAT MADTOM	-	-	-	-	-
WHITE CRAPPIE	-	-	-	-	-
BLACK CRAPPIE	-	-	-	-	-
ROCK BASS	-	-	-	-	-
SMALLMOUTH BASS	-	-	-	-	-
LARGEMOUTH BASS	-	0.5	-	4.0	-
WARMOUTH SF	-	-	-	-	-
GREEN SUNFISH	1.3	2.0	2.0	31.0	-
BLUEGILL SUNFISH	1.3	0.5	3.5	36.5	-
PUMPKINSEED SUNFISH	-	2.0	4.5	14.0	-
B'GILL X PUMPKINSEED	-	-	-	-	-
GREEN SF X BLUEGILL	-	-	0.5	-	-
GR'N SF X PUMPKINS'D	-	-	1.0	-	-
YELLOW PERCH	-	-	0.5	-	-
BLACKSIDE DARTER	-	-	-	-	-
LOGPERCH	-	-	-	-	-
JOHNNY DARTER	48.7	1.5	0.5	22.0	412.0
GREENSIDE DARTER	44.0	1.0	2.0	9.5	-
BANDED DARTER	-	-	-	-	-
VARIEGATE DARTER	-	-	-	-	-
RAINBOW DARTER	-	-	-	-	-
FANTAIL DARTER	0.7	-	0.5	-	-
FRESHWATER DRUM	-	-	-	-	-
MOTTLED SCULPIN	16.0	0.5	-	-	-
BROOK STICKLEBACK	8.7	4.5	6.0	14.5	111.3
TOTAL RELATIVE NUMBER	1548.7	1075.0	961.0	868.0	2188.0
TOTAL NUMBER SPECIES	19.0	19.0	18.0	16.0	11.0
TOTAL NUMBER HYBRIDS	0.0	0.0	2.0	0.0	0.0

Table 4. Organisms collected from artificial substrate samplers from Middle Fork Little Beaver Creek, August 14–September 26, 1985[a].

Taxa	Station (RM)[b] 40.3	38.3	37.7	36.7	35.4
Annelida:					
Oligochaeta	16	+	62+	664+	64+
Helobdella stagnalis				4+	3+
Helobdella triserialis					114+
Placobdella ornata			+		
Erpobdella punctata					+
Crustacea:					
Asellus sp	+		+	132+	80+
Crangonyx sp					64+
Hyalella azteca	+		+	+	2176+
Orconectes obscurus	1+				+
Ephemeroptera:					
Baetis sp	+				
Calibaetis sp				+	
Cloeon sp	+				
Stenacron sp	+				
Stenonema pulchellum	+				
Odonata:					
Calopteryx sp		+			
Coenagrionidae	4	+	+	+	16+
Hemiptera:					
Microvelia sp	+	+	+		
Belostoma sp				+	+
Sigara sp	+		+	+	+
Notonecta sp			+		+
Megaloptera:					
Sialis sp	+				
Chauliodes sp			+		
Trichoptera:					
Cheumatopsyche sp	+	+			305+
Hydropsyche depravata group	+	+			23+
Symphitopsyche bifida group	+				8
Symphitopsyche slossonae		+			
Coleoptera:					
Peltodytes sp			+	+	+
Hydroporus sp			+		
Ilybius sp			+		
Laccophilus sp			+	+	
Cymbiodyta sp					+
Enochrus sp	+				

Table 4. (Continued).

Taxa	Station (RM)[b] 40.3	38.3	37.7	36.7	35.4
Helophorus sp				+	
Paracymus sp		+			
Tropisternus sp		+			+
Ectopria nervosa					+
Dubiraphia sp				1	
Optioservus sp	+				
Optioservus fastiditus		+			
Stenelmis sp	+	+		+	
Diptera:					
Limonia sp			+		
Tipula sp	+		+		
Tipula abdominalis		+			
Anopheles sp	+		+		
Culex sp			+		
Simulium sp	+	+			37+
Ablabesmyia parajanta	14				
Conchapelopia sp		+			99+
Helopelopia sp	+	+			
Larsia sp	14			12	
Meropelopia sp					99
Natarsia sp A			320+	143	
Procladius sp	14+	+		+	
Thienemannimyia sp					298
Cricotopus bicinctus					+
Cricotopus tremulus group					+
Cricotopus trifascia group					99+
Parametriocnemus sp	+				
Thienemanniella prob. xena					198+
Tvetenia bavarica	+				
Chironomus decorus group	164+		27+	95+	+
Chironomus riparius group			222+	+	
Cryptochironomus fulvus group	+			+	
Dicrotendipes neomodestus	164				
Dicrotendipes nervosus Type I	14			59	
Dicrotendipes nervosus Type II	55		257+	618	+
Kieferrulus dux				+	
Microtendipes pedullus group	492+				
Phaenopsectra sp		+			
Polypedilum (P.) convictum					2088+
Polypedilum (P.) fallax group	14	+			99+
Polypedilum (P.) illinoense		+	9		99+

Table 4. (Continued).

Taxa	Station (RM)[b] 40.3	38.3	37.7	36.7	35.4
Polypedilum (Tripodura) nr. scalaenum					+
Stictochironomus sp	14				
Paratanytarsus sp	328			380	5368+
Rheotanytarsus exiguus group	+				298
Tanytarsus sp	82				
Tanytarsus glabrescens group	14				
Tanytarsus guerlus group					99+
Limnophora aequifrons	+	+			
Mollusca:					
Physella sp	2+	+	+	2+	174
Helisoma anceps	+				
Planorbella pilsbryi	10+				17+
Ferrissia sp	3				
Number of organisms/sq. ft.[c]	283.8		179.4	422	2385
Total number of quantitative taxa	19		6	11	24
Total number of qualitative taxa	34	21	22	18	33
Diversity index $\bar{d}$[c]	2.80		2.03	2.42	2.61

[a] Qualitative samples were collected from natural substrates and the taxa collected are indicated by a +.

[b] RM (river mile).

[c] Artificial substrate sample only.

Table 5. Organisms collected from artificial substrate samplers from Middle Fork Little Beaver Creek, August 14–September 26, 1985[a].

Taxa	Station (RM)[b] 32.6	28.8	26.9	25.1	24.8	21.8
Coelenterata:						
Hydra sp		48		16		32
Platyhelminthes:						
Turbellaria	21	52+				
Bryozoa:						
unidentified	+					
Annelida:						
Oligochaeta	305	64	32	40		56+
Helobdella stagnalis	+					
Helobdella triserialis	+					
Microstoma fervida			+			
Crustacea:						
Asellus sp	2+	4+	+	+		
Crangonyx sp	12	5	+			1+
Hyalella azteca	106+	+	+	+	+	1+
Orconectes obscurus		1		+	+	
Ephemeroptera:						
Isonychia sp						1
Baetis sp	70+		46+		+	367+
Centroptilum sp		1				
Stenacron sp	1	4+	7+	12+	+	6+
Caenis sp	207+	52+	7	16+		+
Odonata:						
Calopteryx sp	1+	4+	26+	+	+	6+
Coenagrionidae	1+	2+	+	+		
Argia sp		+	+			
Basiaeschna janata	+	+				
Hemiptera:						
Microvelia sp						+
Rhagovelia sp			+			
Belostoma sp						+
Rantra sp		+		+		
Pelocoris sp		+				
Sigara sp		+				+
Trichocorixa sp		+				
Notonecta sp		+				
Trichoptera:						
Lype diversa				4		88
Cheumatopsyche sp	102+		375+		+	649+
Hydropsyche depravata	8+		6+	+	+	115+
Symphitopsyche bifida group	48+	+			+	714+
Symphitopsyche slossonae						14

Table 5. (Continued).

Taxa	Station (RM)[b] 32.6	28.8	26.9	25.1	24.8	21.8
Hydroptilidae	+					
Hydroptila sp	79	17				12
Coleoptera:						
Hydroporus sp		+				
Tropisternus sp	+					+
Ancyronyx variegata		+				
Dubiraphia sp	+					40
Dubiraphia vittata		2				+
Macronychus glabratus	+	1+	30+			1
Optioservus sp		+				
Optioservus fastiditus					+	
Stenelmis sp	13+		26+		+	1+
Diptera:						
Tipula sp						+
Tipula abdominalis			1+		+	
Anopheles sp		+				
Simulium sp	43+		17+		+	+
Ceratopogonidae		16	16			
Ablabesmyia mallochi		79		36		
Conchapelopia sp	503+	516	236	54		129+
Labrundinia pilosella		40				
Labrundinia Type II		40+				
Nilotanypus sp			118			
Procladius sp		40+	+	18		
Thienemannimyia sp		40	236	18		388
Corynoneura taris	251	40	59			
Cricotopus sp						388
Cricotopus bicinctus					+	2071+
Cricotopus tremulus group	126		177		+	3365+
Cricotopus trifascia group					+	
Limnophyes sp		+				
Nanocladius sp		40				
Nanocladius distinctus			177			129
Parakiefferiella Type I		376+	118	161		
Parakiefferiella Type II		159		18	+	518
Parametriocnemus sp			+	18		
Rheocricotopus prob. robacki	63+		177		+	1165
Thienemanniella prob. xena			59			
Chironomus decorus group	63+		+	18+		
Chironomus riparius group			+	196	+	+
Dicrotendipes neomodestus	629+	991+	59	107		

Table 5. (Continued).

Taxa	Station (RM)[b] 32.6	28.8	26.9	25.1	24.8	21.8
Dicrotendipes nervosus Type I		476+		71		
Dicrotendipes nervosus Type II	126	635+		107		
Microtendipes pedullus group			59			
Paralaterborniella nigrohateralis		+				
Paratendipes sp	+					
Phaenopsectra sp	+					
Phaenopsectra prob. dyari					+	
Polypedilum (P.) convictum	252+	+				129
Polypedilum (P.) fallax group	377+	119	118+	89		
Polypedilum (P.) illinoense			+			
Polypedilum (Tripodura) nr. scalaenum	126		177	36		
Stictochironomus sp			+	36+	+	
Paratanytarsus sp	3521+	278	354	268+		
Rheotanytarsus exiguus group	565		2595			647+
Tanytarsus sp	503+	674	+	71		
Tanytarsus glabrescens group		367	531	18		
Tanytarsus guerlus group	64	+		36		
Chrysops sp	+					+
Empididae	64	16	146			1040
Mollusca:						
Physella sp	37+	1	+	+	+	+
Ferrissia sp	+	1	49			12
Pisidium sp					+	
Sphaerium sp	+	+	+	+		
Number of organisms/sq. ft.[c]	1658	1039	1207	293		2417
Total number of quantitative taxa	32	35	30	24		29
Total number of qualitative taxa	33	31	26	16	22	25
Diversity index $\bar{d}$[c]	3.33	3.72	3.35	3.94		3.40

[a] Qualitative samples were collected from natural substrates and the taxa collected are indicated by a +.

[b] RM (river mile).

[c] Artificial substrate sample only.

Table 6. Organisms collected from artificial substrate samplers from Middle Fork Little Beaver Creek, August 14–September 26, 1985a.

Taxa	Station (RM)b				
	20.8	15.1	10.9	9.0	1.9
Porifera:					
Eunapius fragilis			+		
Coelenterata:					
Hydra sp	8			16	32
Platyhelminthes:					
Turbellaria		+	+		2
Annelida:					
Oligochaeta	249+	57+	2688+	336	280
Crustacea:					
Asellus sp		+	36+	9+	+
Crangonyx sp	+	+	+	+	+
Hyalella azteca	+		+		
Orconectes obscurus	+	+	+	+	+
Ephemeroptera:					
Isonychia sp	1	2+	2+	1	81+
Baetis sp	43+	24+	13+	2	2+
Stenacron sp	10	480+	5+	39+	115+
Stenonema pulchellum		133+	35+	9+	527+
Stenonema terminatum					61
Stenonema tripunctatum	6				
Stenonema vicarium	15	+			7
Paraleptophelebia sp	1	1			
Tricorythodes sp			2	1	12
Caenis sp	101	271+	86+	13+	328+
Ephemeridae				16	
Ephemera simulans		1			12+
Odonata:					
Calopteryx sp	5	2+	1	2+	
Hetaerina sp				1	
Coenagrionidae	3+		+	17+	8+
Argia sp				+	+
Boyeria vinosa		+			+
Gomphidae			1		1
Ophiogomphus sp				+	+
Plecoptera:					
Pteronarcys sp					1+
Perlodidae					16
Hemiptera:					
Microvelia sp		+			
Sigara sp	+				

Table 37. Violations of Ohio EPA Warmwater or Exceptional Warmwater Habitat water quality standards (OAC 3745-1) for chemical/physical parameters measured in the Little Beaver Creek study area, 1985.

Stream Name	River Mile	Violation: Parameter(value)[a]
Middle Fork Little Beaver Creek	40.3	Dissolved Oxygen (2.9), Fe (2780)
	38.3	Fe (4170, 1280, 8070, 1670)
	37.7	Dissolved Oxygen (3.0, 2.8, 0.7, 1.3, 2.5, 2.4, 1.0, 2.0), Fe (3740, 1260), Ammonia-N (13.2, 15.5, 13.0, 16.4)
	36.7	Dissolved Oxygen (2.0, 3.5, 1.1, 2.7, 3.1, 2.0, 1.5, 3), Ammonia-N (14.6, 16.5, 13.9, 18.0)
	35.4	Dissolved Oxygen (3.0, 1.9, 3.4, 1.7), Fe (5860), Ammonia-N (11.3, 11.3, 9.35, 15.5)
	32.0	Dissolved Oxygen (2.8), Fe (9300, 1980), Ammonia-N (4.05)
	28.8	Fe (4550), Ammonia-N (3.08)
	27.8	Fe (1250, 1230)
	25.1	Fe (2110, 1840)
	21.8	Fe (3020, 1660, 1640, 1660, 1770)
	20.9	Fe (2260, 2180, 1830, 2470)
	15.1	Fe (1600)
	11.0	Fe (1290, 1400)
	9.0	Fe (1390)
North Fork Little Beaver Creek	7.3	Phenol (10)
	5.6	Zinc (830)
West Fork Stateline Creek	1.5	Fe (1600, 2940, 1870, 2000, 2080)
Leslie Run	3.3	Copper (11.4, 68.5, 61, 30), Iron (1240, 1670), Zinc (315, 1830, 13,000, 5320, 30,000), Ammonia-N (6.35, 5.75, 4.05, 16), Phenolic (10)
	1.9	Copper (40.4), Iron (2630), Zinc (1320, 1290, 890, 4110, 3060), Ammonia-N (6.75)
	0.2	Copper (159), Iron (8,000), Zinc (540, 345, 470, 9810, 1220), Phenolics (12)
Bull Creek	0.6	Copper (42.8), Iron (2350), Zinc, (3040, 380)
East Branch Middle Fork Little Beaver Creek	0.1	Iron (2140)
West Fork Little Beaver Creek	12.9	Iron (3340, 10,301, 1140)
	12.7	Iron (3090), Phenolics (17)
	9.2	Iron (1210)
	4.1	Iron (1840, 1170, 1300)
	0.8	Iron (11,100)

APPENDIX M

Breeding Bird Populations

APPENDIX M Breeding Bird Populations Breeding Birds in Mahoning and Columbiana Counties (Peterjohn and Rice 1991)		
Species	Status[a] Mahoning County	Status Columbiana County
Pied-billed grebe	P	C
American bittern	C	C
Great blue heron	C	C
Green-backed heron	Pr	Pr
Canada goose	C	C
Wood duck	C	C
American black duck	-	C
Mallard	C	C
Blue-winged teal	-	C
Gadwall	-	P
Turkey vulture	C	C
Northern harrier	C	P
Sharp-shined hawk	C	P
Cooper's hawk	C	C
Red-shouldered hawk	C	P
Broad-winged hawk	C	C
Red-tailed hawk	C	C
American kestrel	C	C
Ring-necked pheasant	C	C
Ruffed grouse	P	C
Wild turkey	-	P
Northern bobwhite	C	C
Virginia rail	Pr	Pr
Sora rail	Pr	Pr
Common moorhen	C	P

APPENDIX M Breeding Bird Populations Breeding Birds in Mahoning and Columbiana Counties (Peterjohn and Rice 1991)		
Species	Status[a] Mahoning County	Status Columbiana County
Killdeer	C	C
Spotted sandpiper	C	C
Common snipe	C	Pr
American woodcock	C	C
Rock dove	C	C
Mourning dove	C	C
Black-billed cuckoo	-	C
Yellow-billed cuckoo	C	C
Barn owl	C	C
Eastern screech owl	C	C
Great horned owl	C	C
Barred owl	C	C
Long-eared owl	C	-
Common nighthawk	Pr	Pr
Wip-poor-will	-	Pr
Chimney swift	C	C
Ruby-throated hummingbird	C	C
Belted kingfisher	C	C
Red-headed woodpecker	C	C
Red-bellied woodpecker	C	C
Downy woodpecker	C	C
Hairy woodpecker	C	C
Northern flicker	C	C
Pileated woodpecker	C	C
Eastern wood-pewee	C	C
Acadian flycatcher	C	C

APPENDIX M Breeding Bird Populations Breeding Birds in Mahoning and Columbiana Counties (Peterjohn and Rice 1991)		
Species	Status[a] Mahoning County	Status Columbiana County
Alder flycatcher	C	Pr
Willow flycatcher	C	C
Least flycatcher	-	Pr
Eastern phoebe	C	C
Great crested flycatcher	C	C
Eastern kingbird	C	C
Horned lark	C	C
Purple martin	C	C
Tree swallow	C	C
Northern rough-winged swalow	C	C
Bank swallow	Pr	Pr
Cliff swallow	-	C
Barn swallow	C	C
Blue jay	C	C
American crow	C	C
Black-capped chickadee	C	C
Carolina chickadee	Pr	C
Tufted titmouse	C	C
Red-breasted nuthatch	-	Pr
White-breasted nuthatch	C	C
Brown creeper	-	Pr
Carolina wren	C	C
House wren	C	C
Marsh wren	C	Pr
Blue-gray gnatcatcher	C	C
Eastern bluebird	C	C

APPENDIX M Breeding Bird Populations Breeding Birds in Mahoning and Columbiana Counties (Peterjohn and Rice 1991)		
Species	Status[a] Mahoning County	Status Columbiana County
Veery	C	Pr
Wood thrush	C	C
American robin	C	C
Gray catbird	C	C
Northern mockingbird	Pr	C
Brown thrasher	C	C
Cedar waxwing	C	C
Logerhead shrike	-	P
White-eyed vireo	C	C
Solitary vireo	-	C
Yellow-throated vireo	C	C
Warbling vireo	C	C
Red-eyed vireo	C	C
Blue-winged warbler	C	C
Golden-winged warbler	-	P
Northern parula warbler	-	Pr
Yellow warbler	C	C
Chestnut-sided warbler	C	Pr
Black-throated green warbler	-	C
Yellow-throated warbler	-	C
Prairie warbler	-	C
Cerulean warbler	C	C
Black-and-white warbler	-	Pr
American redstart	C	C
Prothonotary warbler	-	Pr
Worm-eating warbler	-	C

APPENDIX M Breeding Bird Populations Breeding Birds in Mahoning and Columbiana Counties (Peterjohn and Rice 1991)		
Species	Status[a] Mahoning County	Status Columbiana County
Ovenbird	C	C
Lousiana waterthrush	C	C
Kentucky warbler	C	C
Common yellowthroat	C	C
Hooded warbler	C	C
Yellow-breasted chat	C	C
Summer tanager	-	P
Scarlet tanager	C	C
Northern cardinal	C	C
Rose-breasted grosbeak	C	C
Indigo bunting	C	C
Rufous-sided towhee	C	C
Chipping sparrow	C	C
Field sparrow	C	C
Vesper sparrow	C	C
Savannah sparrow	C	C
Grasshopper sparrow	C	C
Henslow's sparrow	C	C
Song sparrow	C	C
Swamp sparrow	C	C
Bobolink	C	C
Red-winged blackbird	C	C
Eastern meadowlark	C	C
Common grackle	C	C
Brown-headed cowbird	C	C
Orchard oriole	Pr	C

APPENDIX M
Breeding Bird Populations
Breeding Birds in Mahoning and Columbiana Counties (Peterjohn and Rice 1991)

Species	Status[a] Mahoning County	Status Columbiana County
Northern oriole	C	C
Purple finch	C	C
House finch	C	C
American goldfinch	C	C
House sparrow	C	C

[a] Status
C: Confirmed breeding (seven or more males sited, used nests, female with eggs, young fledglings,adult carrying fecal sac, adult with food for young, active nest, identifiable nest with eggs, nest with young)

Pr: Probable breeding (pair observed in suitable breeding habitat, singing males present, territorial behavior observed, courtship displays observed, probable nest site visiting observed, nest building observed)

P: Possible breeding (species observed in breeding season in possible nesting habitat)

-: No observations

APPENDIX N

Mammal Population Information

APPENDIX N Mammal Population Information Mammals in Mahoning and Columbiana Counties (Gottschang 1981)		
Species	Status[a] in Mahoning County	Status in Columbiana County
Virginia opossum	W	W
Masked shrew	C	W
Smoky shrew	W	W
Pygmy shrew	W	W
Short-tailed shrew	C	C
Least shrew	W	W
Hairy-tailed mole	C	C
Eastern mole	W	C
Star-nosed mole	C	W
Little brown bat	W	W
Keen's bat	C	W
Indiana bat	W	W
Silver-haired bat	W	W
Eastern pipistrelle	W	W
Big brown bat	C	W
Red bat	W	W
Hoary bat	W	W
Evening bat	W	W
Cottontail rabbit	W	W
Eastern chipmunk	W	C
Woodchuck	W	W
Gray squirrel	W	W
Fox squirrel	W	W
Red squirrel	W	W
Southern flying squirrel	W	W

APPENDIX N
Mammal Population Information
Mammals in Mahoning and Columbiana Counties (Gottschang 1981)

Species	Status[a] in Mahoning County	Status in Columbiana County
Beaver	C	C
Deer mouse	W	W
White-footed mouse	C	C
Meadow vole	C	C
Woodland or pine vole	W	W
Muskrat	W	W
Southern bog lemming	W	C
Meadow jumping mouse	C	C
Woodland jumping mouse	W	W
Coyote	W	W
Red fox	W	W
Gray fox	W	W
Racoon	C	W
Least weasel	C	C
Long-tailed weasel	W	W
Mink	W	W
Striped skunk	W	W
Whitetail deer	C	C

[a] Status W: County is within the species' Ohio range as determined by Gottschang from standard references.

C: County is the site of confirmed record of the species based upon: Gottschang trapping results; specimens/records observed by Gotschang et al. in museum and university collections; and published records.

APPENDIX O

Ohio Natural Heritage Inventory Data

OHIO
Department of Natural Resources

George V. Voinovich • *Governor*
Frances S. Buchholzer • *Director*

May 18, 1993

Mr. Tom Angus
Environ Corporation
4350 North Fairfax Drive
Arlington, VA 22203

Dear Mr. Angus:

After reviewing our maps and files, I have compiled a list and provided maps for rare, threatened, and endangered plants and animals within a three mile wide corridor along Middle Fork Little Beaver Creek in Mahoning and Columbiana counties, Ohio. Locations marked by a solid dot are exact and those marked by an open circle are accurate to within a square mile. Status codes are explained on an enclosed sheet. A few of the records are from the 1960's, and the years of those records are included for your information. All bird records represent probable or confirmed nesting status.

Little Beaver Creek is a State and National Wild and Scenic River. Middle Fork Little Beaver Creek is designated scenic from the Elkton Road bridge (Elk Run Township Road 901, Columbiana County) to its confluence with West Fork. Information about Ohio's scenic rivers is included for your information. If you need more information, call the scenic river coordinator for this river, Steve Roloson, at (216) 297-7226.

Because our inventory program relies on information supplied by a number of individuals and organizations, a lack of records for any particular area is not a statement that special plant or animal species are absent from a site. Please note that we inventory only high-quality plant communities and do not maintain an inventory of all Ohio wetlands.

Please contact me at (614) 265-6409 if I can be of further assistance.

Sincerely,

Jennifer Hillmer, Ecological Analyst
Division of Natural Areas & Preserves

JH/slc
Enclosures

DNR 0001

Fountain Square • Columbus, Ohio 43224-1387

Ohio Department of Natural Resources
Division of Natural Areas and Preserves

Natural Heritage Elements within a 3-mile Wide Corridor of Middle Fork Little Beaver Creek, Mahoning and Columbiana Counties, Ohio

May 18, 1993

Damascus Quad
Animals

1. Botaurus lentiginosus - American Bittern, E

Salem Quad
Plants

2. Deschampsia flexuosa - Crinkled Hairgrass, T (1967)

Animals

3. Accipiter striatus - Sharp-shinned Hawk, S
4. Porzana carolina - Sora, S
5. Rallus limicola - Virginia Rail, S

Lisbon Quad
Plants

6. Desmodium illinoense - Prairie Tick-trefoil, E (1960)
7. Salix nigra - Black Willow, State Co-champion
8. Phegopteris connectilis - Long Beech-fern, P (1960)
9. Corallorhiza maculata - Spotted Coral-root, P (1964)
10. Glyceria grandis - Tall Manna-grass, P
11. Tritton Marsh
    Carex albolutescens - Pale Straw Sedge, T
    Carex straminea - Straw Sedge, T
    Carex utriculata - Beaked Sedge, P
12. Platanthera flava - Tubercled Rein-orchid, P (1960)
13. Nemopanthus mucronatus - Catberry, P
14. Carex projecta - Necklace Sedge, P
15. Arisaema stewardsonii - Swamp Jack-in-the-pulpit, P
16. Carex straminea - Straw Sedge, T
17. Luzula bulbosa - Southern Woodrush, T (1967)

Animals

18. Porzana carolina - Sora, S
19. Rallus limicola - Virginia Rail, S

Elkton Quad
Managed Areas

20. Beaver Creek State Park. Owned and managed by ODNR Division of Parks and Recreation, 1952 Belcher Drive, C-3, Columbus, OH 43224, telephone (614) 265-6561. See enclosed brochure.

The scenic designation for Middle Fork Little Beaver Creek begins at the Elkton Road bridge. See enclosed brochures for more information.

An explanation of status codes is on a separate sheet.

Division of Natural Areas & Preserves
Ohio Department of Natural Resources

Endangerment Codes

Federal Status Code

FE = Federal endangered
FT = Federal threatened
F1 = Category 1 - F&WS has on file substantial information on biological vulnerability and threat(s) to support the appropriateness of proposing to list these taxa as endangered or threatened species. Proposed rules have not yet been issued because this action is precluded at present by other listing activity.
F2 = Category 2 - F&WS has information which indicates that proposing to list these taxa as endangered or threatened species is possibly appropriate, but substantial data on vulnerability and threat(s) are not currently known or on file to support the immediate preparation of rules.

Ohio Status Codes

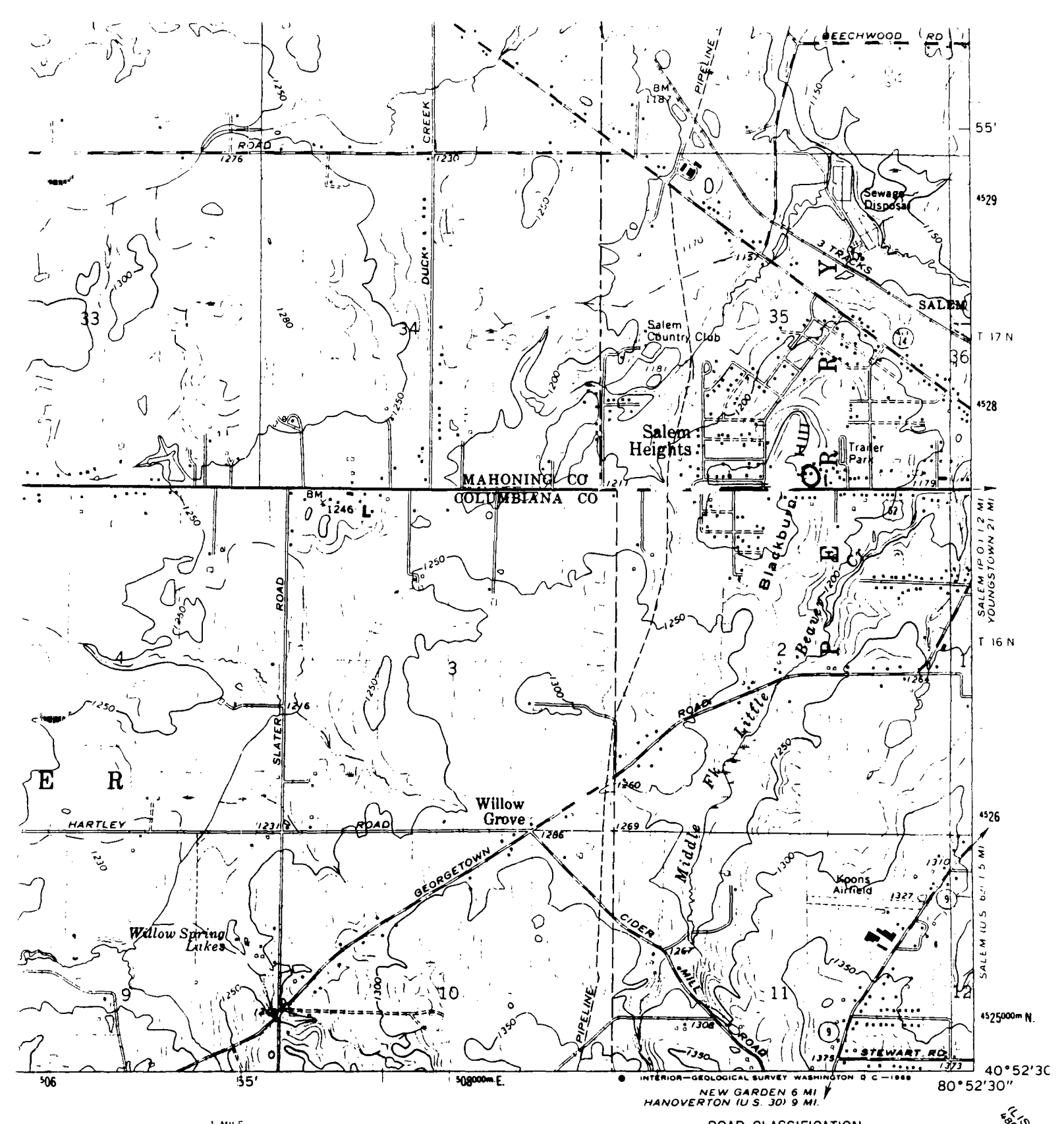
Animals: (assigned by Division of Wildlife)

E = State endangered
T = Threatened (not a legal designation)
S = Special interest (not a legal designation)
X = Extirpated (not a legal designation)

Animals without an Ohio status are included in the Ohio Heritage rare species inventory, but have not been assigned a state status by the Division of Wildlife.

Plants: (assigned by the Division of Natural Areas and Preserves)

E = State endangered
T = State threatened
P = Potentially threatened (Not a legal designation)
A = A species which has been recently added to the Natural Heritage inventory. An endangerment status has not been determined.
X = Presumed extirpated. Has not been collected in Ohio in the last 20 years.



1 MILE

6000 7000 FEET

1 KILOMETER

ROAD CLASSIFICATION

Heavy-duty — Light-duty

Medium-duty — Unimproved dirt

U. S. Route — State Route

OHIO

QUADRANGLE LOCATION

STANDARDS
STON, D. C. 20242
AVAILABLE ON REQUEST

DAMASCUS, OHIO

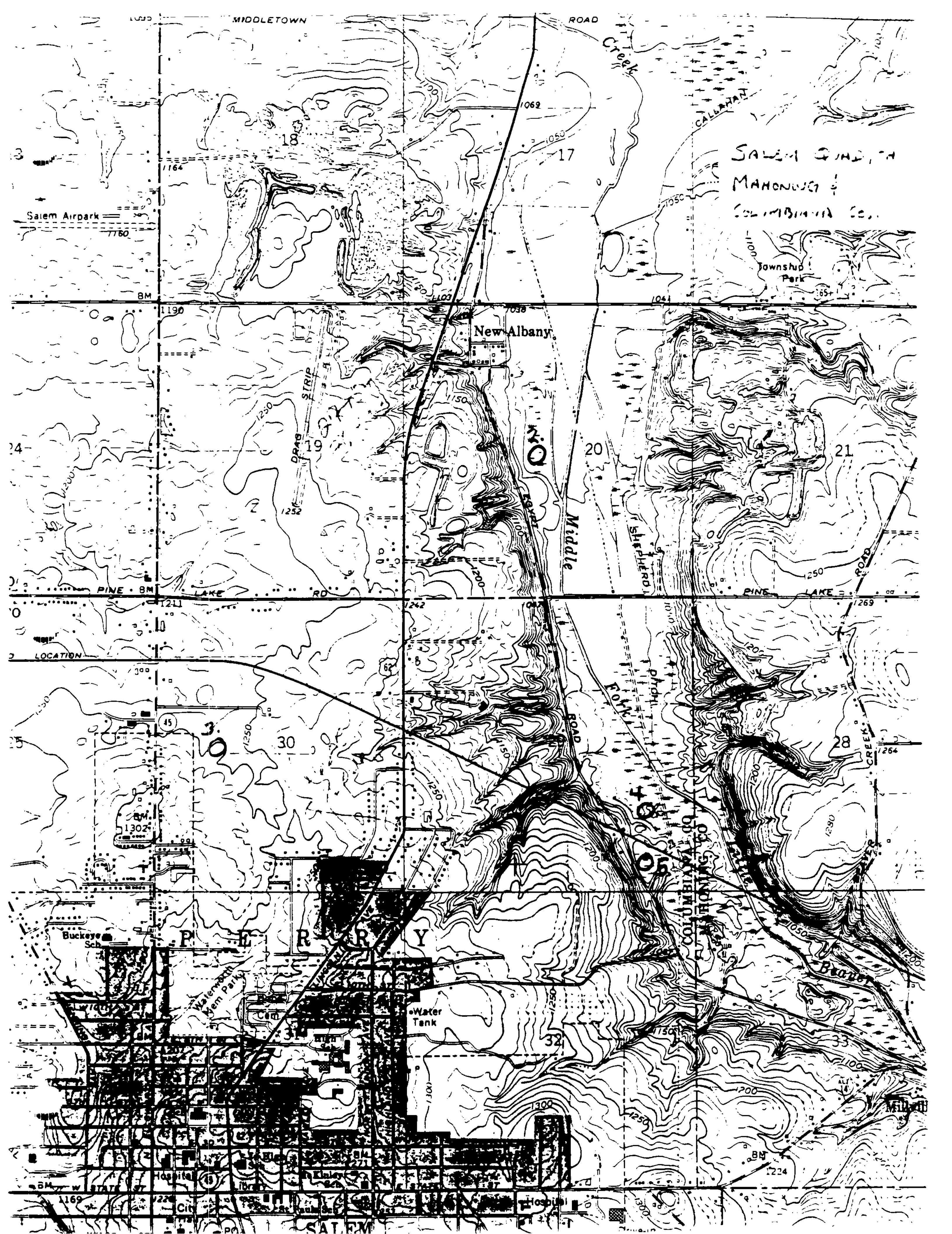
NW/4 LISBON 15' QUADRANGLE

N4052.5—W8052.5/7.5

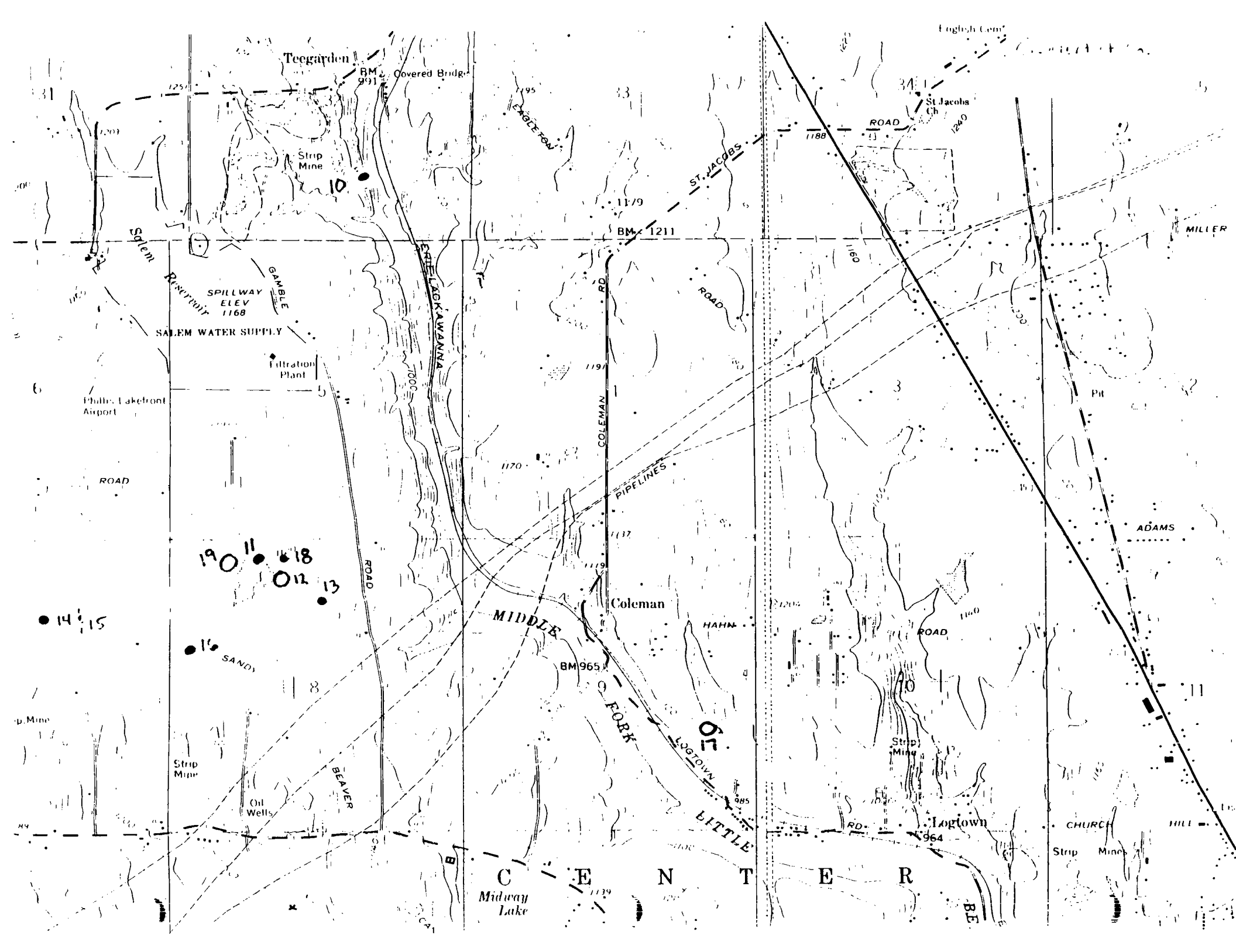
1966

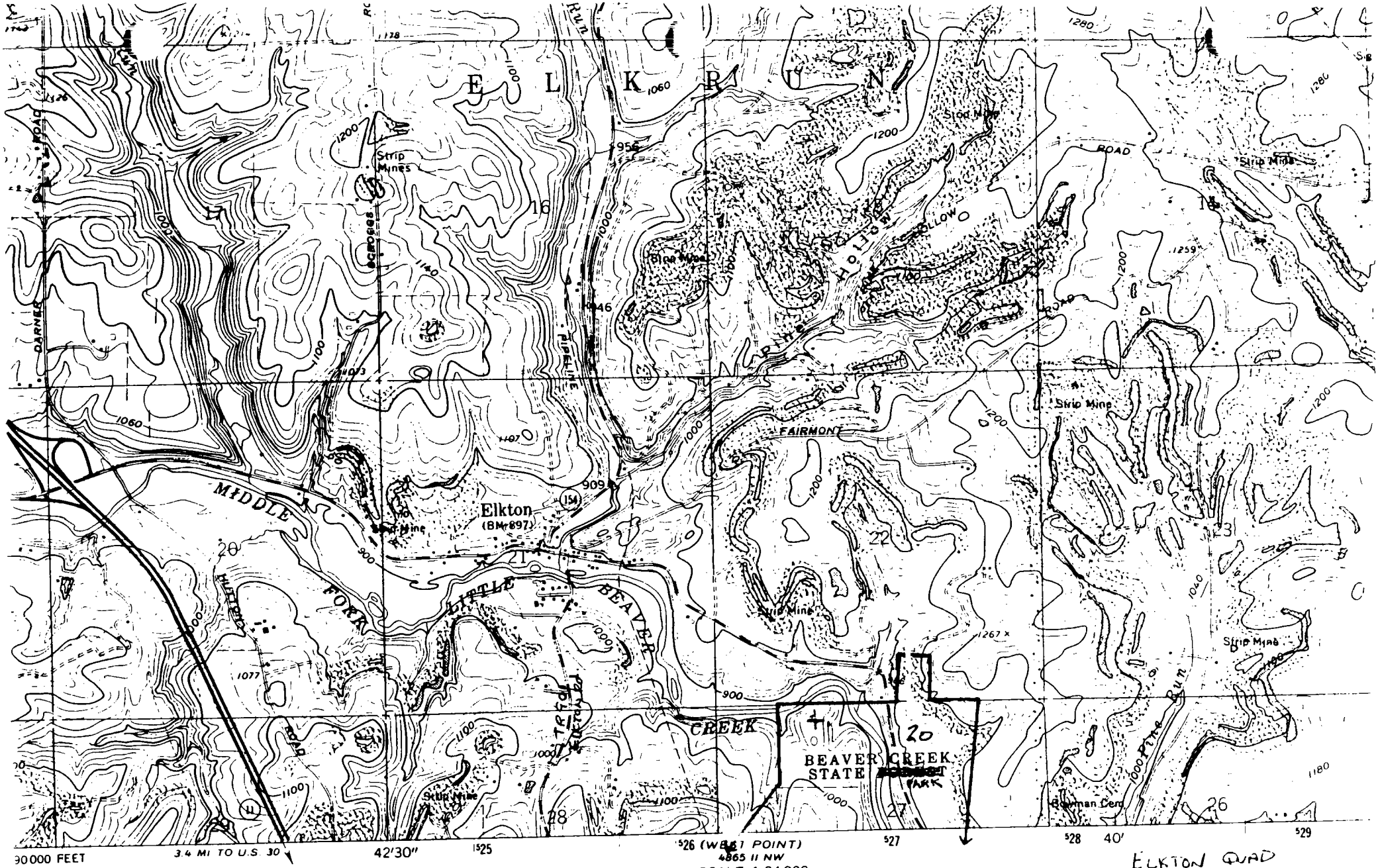
AMS 4865 IV NW—SERIES V852

(LISBON 4865 IV SE)



MIDDLETOWN
ROAD
Creek
CALLAHAN
Salem Airpark
18
17
Township Park
New Albany
DRAG STRIP
19
20
21
Middle
Fork
SHEPHERD
PINE
LAKE
RD
ROAD
LOCATION
30
28
CREEK
COLUMBIANA CO
MAHONING CO
P E R R Y
Buckeye Sch
Waterworth Mem Park
Water Tank
31
32
33
Beaver
Hospital
Library
City
SALEM


Teegarden
BM 901
Covered Bridge
Strip Mine
EAGLE RUN
ST. JACOBS
ROAD
St Jacobs Ch
1188
1240
1179
BM 1211
MILLER
Salem Reservoir
SPILLWAY ELEV 1168
GAMBLE
SALEM WATER SUPPLY
Filtration Plant
CREEK
LACKAWANNA
COLEMAN RD
ROAD
1160
Phillips Lakefront Airport
1191
1170
PIPELINES
Pit
ADAMS
ROAD
Coleman
HAHN
MIDDLE
FORK
LOGTOWN
BM965
ROAD
Strip Mine
Logtown
964
CHURCH
HILL
Strip Mine
Oil Wells
BEAVER
SANDY
CENTER
Midway Lake
BE



ELKTON QUAD

COLUMBIANA Co., OHIO

gical Survey

h zone

APPENDIX P

Environmental Media Sampled by Sample Station

APPENDIX P.1 Environmental Media Sampled by Sample Station for Mirex							
Sample Station	Sediment[1] (μg/kg)	Soil[2] (μg/kg)	Fish Tissue[3] (μg/kg)	Sample Station	Sediment[1] (μg/kg)	Soil[2] (μg/kg)	Fish Tissue[3] (μg/kg)
1	22.7	NS[4]	47.3	18	70.4	NS	660.1
2	17.8	NS	NS	19	137.2	NS	NS
3	24.7	NS	NS	19A[5]	39.3	33.3	NS
4	25.9	NS	NS	19B[5]	122.3	37.6	NS
5	155.8	NS	420.1	20	NS	NS	463.0
6A[5]	83.0	NS	NS	21	46.0	NS	NS
6B[5]	32.9	NS	NS	22	187.8	NS	1002.2
6C[5]	100.4	NS	117.6	23	110.0	NS	6282.9
6D[5]	136.5	NS	NS	24	141.0	NS	NS
7	263.9	NS	270.2	25	90.8	NS	NS
8	NS	NS	1460.5	26	194.9	NS	NS
9	NS	NS	2933.1	27	171.3	368.1	NS
10	1687.4	1031.6	NS	28	NS	NS	490.5
11	568	NS	NS	29	25.1	NS	214.2
12	NS	1960.9	NS	30	NS	NS	36.1
13	558.1	NS	5766.8	31	56.8	NS	NS
14	1202.3	NS	NS	32	47.9	NS	NS
15	152.6	NS	531.8	33	90.2	NS	NS
16	46.9	NS	NS	34	30.6	NS	NS
17	72.8	432.7	NS	35	25.1	NS	170.7
36	NS	NS	NS	45	23.2	NS	88.5
37	38.6	NS	35.0	46	23.9	NS	NS
38	71.6	NS	NS	47	22.8	NS	34.1
39	30.0	NS	77.0	48	23.5	NS	40.6
40	25.5	NS	53.4	49	23.0	NS	45.1

APPENDIX P.1
Environmental Media Sampled by Sample Station for Mirex

Sample Station	Sediment[1] (μg/kg)	Soil[2] (μg/kg)	Fish Tissue[3] (μg/kg)	Sample Station	Sediment[1] (μg/kg)	Soil[2] (μg/kg)	Fish Tissue[3] (μg/kg)
41	24.9	NS	NS	50	23.7	NS	33.3
42	22.4	NS	51.5	51	23.0	NS	26.6
43	29.2	23.3	NS	52	23.0	NS	56.5
44	18.6	NS	71.9				

1 Sediment values are predominantly single samples taken in the Spring of 1990. Occasionally, duplicates were taken.
2 Soil samples represent the mean of four soil samples taken at each of seven sample sites (two on the east bank and two on the west bank).
3 Fish samples are means of at least one set of duplicates, and often include multiple samples representing two trophic levels.
4 NS = not sampled.
5 Samples taken from water bodies connected to MFLBC; not from the creek itself. See RI for explanation.

Appendix P.2 Environmental Media Sampled by Sample Station for 4-Methylphenol	
Sample Station	**Sediment (μg/kg)**
1*	185
2	230
3	1700
13	740
15	2800
42	183
44	2100
*considered background because upstream of site	

APPENDIX Q

Exposure Models

Exposure Models
Exposure Model for Great Blue Heron

$$\frac{(\text{mirex in fish } \mu g/kg)(0.6 \text{ kg/day}) + (\text{mirex in sediment } \mu g/kg)(0.0054 \text{ kg/day})}{3.0 \text{ kg}} = \mu g/kg/day$$

where:0.6 kg/day is the daily fish consumption of a heron (Newell et al. 1987). This conservatively assumes that herons consume entirely fish. Dietary exposure from fish is likely to be less than 100% due to invertebrate, amphibian, reptile, and small mammal components of the diet (Martin et al. 1951),

0.0054 kg/day is incidental soil ingestion rate (9% of diet) using shore bird and goose data from Beyer (1992), and

3.0 kg is the body weight of a great blue heron (Newell et al. 1987)

Exposure Model for Belted Kingfisher

$$\frac{(\text{mirex in fish } \mu g/kg)(0.075 \text{ kg/day})}{0.15 \text{ kg}} = \mu g/kg/day$$

where:0.075 kg/day is the daily fish consumption of a kingfisher (Newell et al. 1987)

0.15 kg is the body weight of a kingfisher (Newell et al. 1987)

Note:Incidental sediment ingestion is judged to be zero due to the feeding habits of this species.

Exposure Model for Sora

$$\frac{(\text{mirex in sediment } \mu g/kg)(BAF_{inv}\ 5.6)(0.021\ kg/d) + (\text{mirex in sediment. } \mu g/kg)(0.004\ kg/d)}{0.071\ kg} = \mu g/kg/day$$

where: 0.021 kg/d is the daily food consumption rate for the sora, considered to consist of primarily aquatic invertebrates,

0.004 kg/d is the daily incidental soil ingestion rate which is calculated from the average (18.08% of dietary intake) of four sandpiper species studied in Beyer (1992),

0.71 kg is the average of body weight data reported in Terres (1980), and

BAF_{inv} 5.6 is the benthic invertebrate bioaccumulation factor calculated as follows:

1. From Connell and Markwell (1990):

$$BAF_{inv} = \frac{\text{bioaccumulation from water}}{\text{partitioning to sediment}}$$

2. From Connell and Markwell (1990):

bioaccumulation from water (BAF_w) = (% lipid content)$(K_{ow})^a$

where: a is a non-linearity constant

partitioning to sediment= organic carbon content(f_{oc}) X K_{oc}

3. Therefore:

$$BAF_{inv} = \frac{(\%\text{lipid content})(K_{ow})^a}{(f_{oc})(K_{oc})} = 5.6$$

given:

a. The lipid content of invertebrates is an average of 15% for 58 genera studied (Hanson et al. 1985);

b. The non-linearity constant (a) for the function describing bioaccumulation of organochlorine compounds from water to invertebrate species is 1.11 (Markwell et al. 1989);

c. The K_{ow} for mirex is 7,762,471;

d. The f_{oc} for a typical sediment is 5%; and

e. The K_{oc} for mirex is 24,000,000.

$$BAF_{inv} = \frac{(0.15)(7,762,471)^{1.11}}{(0.05)(24,000,000)} = 5.6$$

Exposure Model for Virginia Rail

$$\frac{(\text{mirex in sediment } \mu g/kg)(BAF_{inv}\ 5.6)(0.023\ kg/d) + (\text{mirex in sediment. } \mu g/kg)(0.004\ kg/d)}{0.075\ kg} = \mu g/kg/day$$

where: 0.023 kg/d is the daily food consumption rate for the Virginia rail, considered to consist of primarily aquatic invertebrates,

0.004 kg/d is the daily incidental soil ingestion rate which is calculated from the average (18.08% of dietary intake) of four sandpiper species studied in Beyer (1992),

0.75 kg is the average of body weight data reported in Terres (1980), and

BAF_{inv} 5.6 is the benthic invertebrate bioaccumulation factor.

Exposure Model for Robin

$$\frac{(\text{mirex in soil } \mu g/kg)(BAF_{ew}\ 0.51)(0.0087\ kg/d) + (\text{mirex in soil } \mu g/kg)(0.0008\ kg/d)}{0.078\ kg} = \mu g/kg/d$$

where: 0.0087 kg/d is the daily dietary intake of a robin from Levey and Karasov (1989), in this case a conservative assumption that the robin diet is 100% earthworms has been made,

0.0008 kg/d is the incidental soil ingestion rate based on data for woodcock (9.1% of the diet) from Beyer (1992), and

0.078 kg is the body weight of a robin (Levey and Karasov 1989), and

BAF_{ew} 0.51 is the mirex bioaccumulation factor from soil to earthworms and is calculated as follows:

1. From Connell and Markwell (1990):

   $$BAF_{ew} = \frac{\text{bioaccumulation from interstitial soil water}}{\text{partitioning to soil}}$$

2. From Connell and Markwell (1990):

   bioaccumulation from water (BAF_w) = (% lipid content)$(K_{ow})^a$

   where: a is a non-linearity constant

   partitioning to sediment= organic carbon content(f_{oc}) X K_{oc}

3. Therefore:

   $$BAF_{isv} = \frac{(\%\text{lipid content})(K_{ow})^a}{(f_{oc})(K_{oc})} = 0.51$$

   given:

   a. The lipid content of earthworms is 0.85% (Rao and Davidson 1980);

   b. The non-linearity constant (a) for the function describing bioaccumulation of organochlorine compounds from water to whole earthworms is 1.14 (Lord et al. 1980);

   c. The K_{ow} for mirex is 7,762,471;

   d. The f_{oc} assumed for a typical soil is 5%; and

   e. The K_{oc} for mirex is 24,000,000.

   $$BAF_{isv} = \frac{(0.0085)(7,762,471)^{1.14}}{(0.05)(24,000,000)} = 0.51$$

Exposure Model for Red Fox

$$\frac{(\text{mirex in white-footed mouse } \mu g/kg)(0.32\ kg/d)(0.5) + (\text{mirex in vole } \mu g/kg)(0.32\ kg/d)(0.5)}{5.6\ kg} +$$

$$\frac{(\text{mirex in soil } \mu g/kg)(0.009\ kg/d)}{5.6\ kg} = \mu g/kg/d$$

where: 0.32 kg/d is the daily food consumption rate for the red fox (Chapman and Feldhamer 1982),

0.5 is an assumed dietary proportion of 50% white-footed mouse and 50% vole. This conservative assumption attributes 100% of the red fox diet to animal tissue.

0.009 kg/d is the daily incidental soil intake for the red fox (Beyer 1992), and

5.6 kg is the average body weight for the red fox from data in Burt and Grossenheider (1976).

Mirex in white-footed mice is calculated as follows and assumes daily equilibrium between tissues and diet:

$$\frac{(\text{mirex in soil } \mu g/kg)(BAF_{ew}\ 0.51)(0.007\ kg/d)(0.38) + (\text{mirex in soil } \mu g/kg)(BAF_p\ 0.31)(0.007\ kg/d)(0.62)}{0.023\ kg} +$$

$$\frac{(\text{mirex in soil } \mu g/kg)(0.000176\ kg/d)}{0.023\ kg} = \mu g/kg \text{ tissue}$$

where: 0.007 kg/d is the daily dietary intake (32% of body weight) of a white-footed mouse by analogy to the field vole (Chapman and Feldhamer 1982),

0.38 and 0.62 are the average proportions of the diet for animal and vegetative matter, respectively (Martin et al. 1951),

0.023 kg is the body weight of a white footed mouse from data in Burt and Grossenheider (1976),

BAF_p 0.31 is the average mirex bioaccumulation factor from field soil to the above-ground portions of four species of plants (de la Cruz and Rajanna 1975), and

BAF_{ew} 0.51 is the mirex bioaccumulation factor from soil to terrestrial earthworms.

Mirex in voles is calculated as follows and assumes daily equilibrium of mirex between tissues and diet:

$$\frac{(\text{mirex in soil } \mu g/kg)(BAF_p\ 0.31)(0.0165\ kg/d) + (\text{mirex in soil } \mu g/kg)(0.00038\ kg/d)}{0.049\ kg} = \mu g/kg \text{ tissue}$$

where: 0.0165 kg/d is the daily dietary intake (32% of body weight) of a field vole (Chapman and Feldhamer 1982),

0.049 kg is the body weight of a file vole from data in Burt and Grossenheider (1976), and

BAF_p 0.31 is the average mirex bioaccumulation factor from field soil to the above-ground portions of four species of plants (de la Cruz and Rajanna 1975), and
0.0038 kg/d is the incidental soil ingestion for the meadow vole (Beyer 1992).

Exposure Model for Northern Harrier

$$\frac{(\text{mirex in white-footed mouse } \mu g/kg)(0.0745\ kg/d)(0.5) + (\text{mirex in vole } \mu g/kg)(0.0745\ kg/d)(0.5)}{0.521\ kg} = \mu g/kg/d$$

where: mirex in mouse and vole are calculated as in the red fox model,

0.0745 kg/d is the average daily dietary intake for the norhern harrier from data in Craighead and Craighead (1969),

0.5 is an assumed dietary proportion for white-footed mice and field voles. that conservatively ssumes that 100% of a northern harriers diet is from white-footed mice and field voles, and

0.521 kg ia the avrage body weight for northern harriers from data in Craighead and Craighead (1969).

Exposure Model for Mink

$$\frac{(\text{mirex in fish } \mu g/kg)(0.15 \text{ kg/d})(0.46) + (\text{mirex in mouse } \mu g/kg)(0.15 \text{ kg/d})(0.5)(0.36) +}{1 \text{ kg}}$$

$$\frac{(\text{mirex in vole } \mu g/kg)(0.15 \text{ kg/d})(0.5)(0.36) + (\text{mirex in sed. } \mu g/kg)(BAF_{inv}\ 5.6)(0.15 \text{ kg/d})(0.18)}{1 \text{ kg}} = \mu g/kg/d$$

where: 0.15 kg/d is the daily dietary consumption of the mink from data in Bleavins et al. (1980),

0.46, 0.36, and 0.18 are the assumed proportions of mink diet for fish, small mammals, and aquatic invertebrates as calculated from data in Chapman and Feldhamer (1982) and conservatively assuming that these three components comprise 100% of a mink diet.

0.5 is the assumed proportion of the small mammal diet that is mice or voles,

BAF_{inv} 5.6 is the bioacccumulation factor for mirex in sediment to aquatic invertebrates, and

1 kg is the average body weight of a mink from Bleavins et al. (1980).